The American Journal of Surgery

NEW SERIES, VOLUME XLV

JULY TO SEPTEMBER

1939

CONTENTS OF NEW SERIES, VOLUME XLV
ORIGINAL ARTICLES

Contents *v*

EDITORIAL

IS TOTAL PNEUMONECTOMY JUSTIFIED IN CANCER OF THE LUNG?

THE tendency of history to repeat itself is being illustrated today by the wave of popularity which total pneumonectomy is enjoying as a cure for cancer of the lung. Whenever a new field of surgery has been opened up by the removal of some bugaboo, an era of radical procedures in that field has followed. This was true of abdominal surgery when Lister removed the fear of sepsis. Now that fear of alterations in thoracic pressure has been largely removed from chest surgery, total pneumonectomy is being advocated for cancer of the lung, and about fifty cases have been reported within the last five years, attracting wide attention.

It is a sad commentary on the surgical profession that we sometimes become so impressed by the skill and drama of a new surgical technique that, in our enthusiasm to try it, we allow our judgment to be biased as to what is for the best interest of the patient. And when a procedure is advocated by prominent surgeons, others do not always pause to weigh its value in the light of past experience in other fields. Furthermore, when a great surgeon and teacher has stated a clean cut principle, the influence of that teaching is likely to survive even when subsequent experience brings new perspective. Halsted's teaching of radical surgery for all cancer was so thorough-going that radical surgery is still receiving credit for many five year cures which, in the light of our present knowledge of the histology and cytology of various grades of cancer, seems scarcely warranted. The investigations of Von Hansemann, Broders, MacCarty and others all point to the fact that the cellular elements of the particular neoplasm, rather than radical surgery for its removal, determine the ultimate prognosis, particularly with regard to metastasis. The laboratory observations are confirmed by clinical experience.

Certainly there is little in our experience with cancer of

1

the cervix and cancer of the breast, two surgical fields in which we have the advantage of relatively long perspective, to prove that radical surgery is a cure for cancer, especially in high grade cancers. In the early days of treating cancer of the cervix surgically, everyone regarded radical surgery as the operation of choice. But as late end results began to be checked, it was found that the life of a patient with a high-grade neoplasm was short, in spite of radical surgery; and that in Grades I and II the patients did equally well under less mutilating procedures. In other words, the lesson was learned that, so far as this anatomic region was concerned, radical surgery did not prevent metastasis in high grade neoplasms. Women's sexual apparatus were no longer sacrificed wholesale, and today complete hysterectomy, oöphorectomy, and salpingectomy are relatively uncommon.

Two decades ago cancer of the breast was treated with equal radicalism, the axillary glands being removed in most cases. The influence of Bloodgood and Greenough's advocacy of extensive sacrifice of tissue was widespread. How much unnecessary suffering this brought to women with low grade tumors, it is difficult to estimate. Suffice it to say that in many cases today simple mastectomy is giving as good results as radical mastectomy with removal of all glands, for the obvious reason that in the Grade IV tumors it is practically impossible to prevent metastasis. Granting the value of early diagnosis, a clinically "early" case is quite likely to be biologically "late." And it is the biology which will determine the prognosis.

A survey of the literature in these and other surgical fields leads one to believe that the great bulk of cases reported as five-year cures following radical surgery are really low-grade neoplasms whose cells are well differentiated and from which the danger of metastasis is therefore limited. Personal experience confirms this point of view. The good results should be attributed to the relatively benign cellular structure, and not to radical surgery, it being highly probable that less radical surgery would have produced equally good results with less suffering to the patient and less mutilation, with its undoubted psychologic effect.

Is there any reason why the same principles should not hold true in cancer of the lung? Is it not our duty to the patient to profit by experience with cancer in other regions of the body; or must we learn over again, at the expense of the patient's suffering, the lesson that radical surgery does not cure high grade cancer nor materially delay metastasis? As Horsley has recently said in advocating less routinely radical procedures for carcinoma of the upper rectum, "something more than mere existence should be included in the objectives of surgery." Granting that there are occasional cases in which for technical reasons total pneumonectomy must be done, ordinarily by every sound principle of surgery, healthy tissue should be preserved, whenever possible. There is double reason for doing this in the case of the lung, for should the patient develop pneumonia after operation, or subsequently, he is going to be much better off with one and one-half lung thans with only one.

In July, 1936, Walker collected forty-four cases of pneumonectomy, twenty of whom had died, a mortality of slightly less than 50 per cent. Nineteen of the forty-four cases were for cancer; the others, for inflammatory conditions. Of the nineteen for cancer, twelve died shortly after operation, and a thirteenth reported as "recovered" died six months later of recurrence. This gives a mortality rate of 68 per cent for the cancer cases. Walker comments significantly: "How permanent as regards the question of recurrence the results [of total pneumonectomy] will be, can only be ascertained after a much longer experience, but the supervention of both local recurrence and cerebral metastases has already been recorded in cases where it had been hoped that the disease had been entirely

eradicated by complete removal of the lung."

Monod, a French surgeon, writes that "pneumonectomy is apparently more frequently indicated in America," and wonders whether a simpler operation and one less fatal might not gradually gain the confidence of the general practitioner and the patient. "It is not the legitimacy of total pneumonectomy which one argues about, but its efficacy."

Lobectomy will give the patient as good prognosis in low grade neoplasms as will total pneumonectomy; and in high grade neoplasms, experience in other surgical fields gives no promise that radical surgery will prevent metastasis. It seems, therefore, that we should take serious pause before advocating total pneumonectomy as the operation of choice. Lobectomy is biologically more logical.

EDWIN JOSEPH GRACE, M.D., F.A.C.S.

CHARLES MAYO

1865–1939

THE entire country was shocked by the untimely death of Charles Mayo. Newspapers from coast to coast published columns concerning his career. To most members of the medical profession the salient facts of his personal history and of the great clinic founded by his brother and himself are well known. An outline of his biography is unnecessary. The most fitting memorial to his useful life is the great institution which bears his name, whence scientific achievements, revolutions in administration and refinements in techniques have poured forth.

We can simply say that Charles Mayo will be missed. He was a great man and a splendid type of American physician. We feel that with his passing an era in American medicine has ended.

T. S. W.

ROBERT V. DAY

ROBERT V. DAY, prominent urologist and member of the Editorial Board of The American Journal of Surgery, died after a short illness on April 29, 1939. His was a full and interesting life. His friends were legion. He will be missed by hundreds of American physicians, but the loss will not be felt more keenly than by the Editor, Publisher and readers of this Journal.

T. S. W.

CARCINOMA OF THE UTERINE CERVIX*

J. WILLIAM WHITE, M.D.

Director of Tumor Clinic, St. Mary's Hospital

SCRANTON, PENNSYLVANIA

NOWHERE in the history of medicine is there a coördinated effort to control a particular disease comparable to the concerted endeavor to control cancer. Nevertheless the mortality from carcinomatous disease is enormous and its ravages, instead of being diminished, are actually on the increase.

In the past forty years the mortality rate of cancer has doubled; and, during the past five years, there has been an annual rise in the recorded cancer deaths of about forty per million of population.

The aggregate loss of life from this disease in women is appalling, for of all women who die between the ages of 45 and 65 not less than 20 per cent die of carcinoma. Of these, approximately one-third die of carcinoma of the uterus which begins in 90 per cent of cases as carcinoma of the cervix.

Exacting its toll of 10,000 victims annually in the United States,[11] carcinoma of the cervix constitutes a problem of national interest that challenges medical erudition. But despite the inadequacy of current knowledge for the complete suppression of the disease, there are well established facts and sound working principles sufficient to save many lives. The final solution of existing perplexities cannot be awaited; advantage must be taken of knowledge already acquired. The substance of that knowledge must be disseminated repeatedly until comprehended by every physician who deals with cancer.

ETIOLOGY

Although essentially a disease of middle life, 35 to 55 years, carcinoma of the cervix may occur at virtually any age. Probably the youngest patient on record with cervical carcinoma was a child 22 months old whose case Scheffey and Crawford reported.[34]

The direct cause of malignancy of the cervix, as elsewhere, is unknown. There are two main factors accepted as responsible for the development of cancer, viz., an extrinsic factor of chronic irritation, a stimulant to cytogenesis, and an intrinsic factor of susceptibility.[12] It has been estimated that 20 per cent of civilized human beings are predisposed, or exhibit an intrinsic susceptibility to cancer (Willy Meyer).

The uterine cervix is so often the site of chronic infection, erosions, retention cysts, endocervicitis, polyps and lacerations that it is difficult to understand why it does not more often become carcinomatous unless credence is placed in the theory of intrinsic susceptibility. It is impossible to prove any hereditary influence clinically. It appears that a degree of chronic irritation harmless to one individual is sufficient to produce carcinoma in another who, therefore, must be susceptible.

Erosions, circumoral cervicitis (Boller) occur in 75 per cent of parous and 25 per cent of nulliparous women. Cancer of the cervix develops in 97 per cent of cases in

parous women and in the remaining 3 per cent it occurs in the nulliparous with cervical erosions of infectious origin.[32] Bland[6] believes that injury of the cervix during childbirth "forms the propitious background for the ultimate development of cervical cancer."[30]

PATHOLOGY

Carcinoma of the cervix is recognizable histologically either as epidermoid carcinoma or as adenocarcinoma. The epidermoid form may arise in any part of the cervical canal or from the vaginal portion of the cervix about the external os. It is difficult to be certain of the site of growth origin, either grossly or histologically, except in the rarely discovered earliest stages.

Cancer arising in the cervical canal proper may extend centrally to form the so-called inverting type of growth, or it may project distally into the vagina where it presents as an everting, villous-like mass. When it originates about the external os it shows a tendency to spread first to the neighboring vaginal mucous membrane and then to involve the cervical canal secondarily.

Carcinomata with distinct infiltrative qualities that begin at or within the external os may not present the usual appearances of malignancy. Puckering and retraction of the cervix, which, if the process is advanced, involve the vaginal fornices and result in characteristic induration, are suggestive evidence of an infiltrating lesion.

The earliest demonstrable cancerous lesions of the cervix are said to appear as small, leucoplakia-like areas; minute bosselated or papillary projections; small, bleeding, superficial ulcerations; or as localized cervical enlargements due to tumor formation within the wall of the cervix without the loss of surface epithelium.[27]

However, probably because of the mildness and paucity of symptoms, patients rarely seek professional consultation in the earliest stages of the disease. Consequently,

able clinicians of broad experience have gone through a normal span of active practice without an opportunity to study one of these early lesions. Nevertheless it should be remembered that cancer of the cervix manifests itself in its incipience, when it is most amenable to proper treatment, by characteristic features which should be unremittingly contemplated during every vaginal examination.

There are three forms of the disease frequently encountered, which represent later stages of growth:

1. The commonest and most malignant form gives rise to widespread infiltration of the surrounding tissues and may quickly involve the lymph nodes. It extends deeply in the direction of the internal os, causes enlargement of the cervix as a whole and eventually produces a deep crater lined with an irregularly granular, friable, sloughing debris which bleeds readily.

2. A superficial, large, fungating, cauliflower-like mass projects into the vaginal vault where it produces contact growths. It arises in the region of the external os and shows little tendency to invade the deeper structures. It is of intermediate malignancy, although ulceration and hemorrhage occur readily and early.

3. The least common variety occurs as a flat, indurated ulcer of comparatively slow growth which tends to spread superficially.[8]

Carcinoma of the cervix is so multifarious in its variations that it is impossible to describe all of its diversities. Biopsy specimens frequently will reveal areas of malignancy that are otherwise totally unsuspected. When these microscopic areas are examined, the cervical epithelium will be seen to undergo an abrupt hyperplastic change; but the most important alterations are in the epithelial cells proper. There is a loss of normal cell differentiation, and the cells show irregularities in size, shape and staining qualities. Hyperchromatism, anaplasia, and the presence of mitotic figures are observed.

In the absence of invasion, there is hesitancy in some quarters in making a

histologic diagnosis of malignancy. Martz-loff prefers to designate these lesions "probable beginning epidermoid cancer" while Broders favors the term "carcinoma in situ," in the definition of which he includes the intraepidermal epithelioma of Borst and Jadassohn.[9] It remains controversial whether invasion is necessary to a diagnosis of malignancy. There is ample authoritative opinion in support of either side of the question.[29,19] Nevertheless, until the argument is finally settled, it is practical to state that these cell changes should be considered malignant and that each case presenting such changes should be treated accordingly.

Usually carcinoma of the cervix is described as of squamous cell type, but in form it is by no means constant and typical. Variations will be met depending upon whether the growth arises from the squamous epithelium of the vaginal portion of the cervix, or from the columnar epithelium of the cervical canal. Cells representing squamous, transitional and basal layers are encountered, and frequently several types are intermingled in the same neoplasm. In any case, however, there is a predominance of one or another cell type. Not infrequently, because of the cellular characteristics and the degree of stroma response, these cancers are classified as basal, medullary, scirrhous or alveolar forms.

Invasion of the cervix is manifest in broad masses or branching columns of cells, which show many mitotic figures, growing into the deeper structures. The center of these cellular masses may be degenerated and liquefied, a circumstance suggesting the term alveolar carcinoma which is occasionally mistaken for adeno-carcinoma. Changes in the stroma are invariably present, usually in the form of a lymphocytic infiltration along the margin of the tumor. Occasionally the stroma and cells undergo focal hyalinization. Circulatory alterations result in areas of surface necrosis and ulceration that subsequently become infected. On the surface of the neoplasm there are fine, villous-like processes composed of a central blood vessel surrounded by a fibrillary outgrowth of stroma covered with epithelium. These excrescences account for the bleeding which follows slight trauma. When ulceration is extensive, much of the microscopic picture may be obscured by an inflammatory reaction.

Adenocarcinoma, which comprises approximately 5 per cent of cervical cancers, cannot be distinguished grossly from epidermoid carcinoma. However, the microscopic picture is typical in that it presents a glandular structure with more or less intervening fibrous stroma. Adenocarcinoma may arise from glandular epithelium in any part of the cervix; and, although it does not have an invasive tendency, its spread, through metastasis and extension, may closely resemble that of epidermoid carcinoma.

INVASION AND METASTASIS

The rapidity with which a cancer spreads and metastasizes is a fairly constant index of the degree of malignancy of the primary tumor, but it is not an index of its curability at a given period. Considered broadly, the earlier extension and metastasis occur the greater is the degree of malignancy. However, metastasis to the lymph nodes and viscera are not dependent upon the extent of the primary tumor. A very early case may show extreme nodal involvement while another, far more greatly advanced, may produce few or no secondary deposits.

There are three distinct routes by which cancer cells are disseminated:

1. Permeation of connective tissue lymph spaces is the principal source of spread. From the initial site of growth microscopic cell elements extend along these spaces to invade the cellular tissue of the parametrium, or upward through the portio into the supravaginal cervix and thence anteriorly into the vesicovaginal septum with subsequent forward permeation into the bladder wall. They may extend posteriorly into the rectovaginal septum or

pass backward through the lymph spaces of the uterosacral ligaments to invade the rectum. Not infrequently they extend

FIG. 1. Epidermoid carcinoma of differentiated squamous cell type. (× 100.)

downward into the tissues of the vagina, and, less commonly, upward to involve the corpus of the uterus. As the result of necrosis and ulceration various fistulae between the bladder or rectum and the vagina are prone to develop.

2. Metastasis by way of the lymph vessels commonly occurs and may happen during any period of the disease. Often nodes that are nearest the primary tumor, and which naturally are expected to be the first involved, are found absolutely cancer-free while a more distant group will show marked secondary deposits. It would seem that there is a direct relationship between lymph stasis or lymphatic obstruction and the site of neoplasia[23] which, if not of primary etiologic importance, certainly is of respectable significance in the localization of secondary transplants. The order in which groups of

lymph nodes are implicated in carcinoma of the cervix follows: (a) those at the base of the broad ligament; (b) those in the

FIG. 2. Same as Fig. 1. (× 430.)

iliac regions; (c) hypogastric; and (d) sacral groups.

3. Metastasis by way of the blood stream is a late complication of advanced cases. It results in secondary malignant implants in remote tissues, the viscera and bones.

EXTENT OF GROWTH AND PROGNOSIS

Experience conclusively proves that the prognosis in carcinoma of the cervix is primarily dependent upon the extent of the growth and that other considerations, including histologic grading, are comparably less significant.

For the purpose of briefly designating as belonging to one of several groups those cases having common limits of extent, various classifications have been devised. These are based chiefly upon the anatomic distribution of the disease as observed clinically. Most gynecologists have adopted

the clinical classification proposed by the Cancer Committee of the League of Nations, viz.,

Fig. 3. Epidermoid carcinoma of transitional cell type. (× 430.)

Group 1.   Earliest lesions, limited to the cervix; free mobility of the uterus.

Group 2.   Invasion of the vaginal wall; slight involvement of the paracervical and parametrial tissues, uterus mobile.

Group 3.   Marked infiltration of the paracervical and parametrial tissues; uterus fixed.

Group 4.   Invasion of adjacent viscera with or without fistula. May be distant metastases.

Adversity in the prognosis is directly proportional to the progression of the disease from one clinical group to another; and it is also inversely proportional to the resistance of the maturer cancerous cell forms to radiation. The prospect of curability is only further unfavorably altered by complicating factors providing the treatment is adequate and judiciously administered.

Such complicating factors as cardiorenal disease, obesity (Lund) concomitant pregnancy, and a persistent pyometra which affect the plan as well as the results of therapy, together with local infection, the latter universally present but varying in degree and response to treatment, are of contrary influence. Likewise complications arising during the course of therapy, pyelitis, uremia and pelvic peritonitis, any of which may of themselves prove fatal, naturally induce unfavorable effects.

However, when the diagnosis is made early, that is, during the first stage of the disease, cure is said to be possible in approximately 50 per cent of cases,[1] but the probability of cure steadily diminishes as the disease unhappily advances to become a certain fatal malady.[10]

SYMPTOMS

There are no symptoms pathognomonic of carcinoma of the cervix,[2,13] but there are two most important suggestive signs that indicate the possibility of its existence; (a) an unusual discharge, (b) intermenstrual bleeding.

Frequently the principal complaint is of a thin, watery or serous discharge that may be inodorous or malodorous, small in amount or copious. Conversion of an ordinary leucorrhea into a dark brown or hemorrhagic-tinged mucous exudate should lead to the opinion that its source is probably malignant. The sudden yielding of a cervical obstruction, resulting in a gush of serous or purulent material followed by hemorrhage, is a notable sign warranting careful microscopic investigation of curetted cervical tissue.

Most commonly the very important and only symptom which attracts the patient's attention is the unexpected appearance of blood issuing from the genitals. Rarely this bleeding may be in the form of an increased menstrual flow; sooner or later, however, it occurs between periods either without apparent cause or induced by slight trauma, coitus, douching, abdominal effort or defecation. At first the

hemorrhage may be very slight, a few drops characterized as "spotting"; later, however, bleeding occurs more frequently

Fig. 4. Adenopapillary carcinoma. (× 100.)

and in larger quantities. Between profuse hemorrhages in advanced cases there may be a putrid discharge containing necrotic carcinomatous tissue. As the disease advances, recurrent or constant blood loss induces a relative degree of secondary anemia. Toxemia, a later manifestation, results in lassitude, anorexia, fever, sweats and abdominal distention. Pain, never an early complaint, supervenes at the infiltration of neighboring or distant viscera. It, combined with anemia and cachexia, initiates the triad of despair.

DIAGNOSIS

The early diagnosis of cervical cancer is based entirely upon objective findings. It is imperative, therefore, that a critical physical examination be made in every suspected case.

Inspection of the external genitalia may reveal evidence of birth injuries, perineal

relaxation and cicatrices, or the presence of a simple, putrid or hemorrhagic discharge. These signs, although not prime

Fig. 5. Same as Fig. 4. (× 430.)

factors in the diagnosis of malignancy, bear a degree of importance not to be disregarded.

Digital examination is made to determine whether the cervix is smooth, lacerated, soft, cystic, firm, nodular, swollen, indurated or retracted. In early cases palpation may not be informative. Small, superficial ulcerations are not palpable. The presence of friable tissue which bleeds freely when manipulated is characteristic of cervical carcinoma. The existence of vaginal involvement is evident in the palpation of brawny, irregular, local induration.

Bimanual examination reveals the presence or absence of uterine mobility, the presence or absence of induration in the fornices and, to some extent, the degree of involvement of the broad ligaments in later cases.

Rectal examination proves to be unenlightening except in advanced cases. However, vaginorectal examination, a procedure

wherein the index finger is passed into the vagina simultaneously with the insertion of the adjoining mid-finger into the rectum

of the cervix, speculum observation is of no assistance.

In the more advanced infiltrating or

FIG. 6. Adenocarcinoma. (× 100.)

FIG. 7. Same as Fig. 6. (× 430.)

for the purpose of palpating the paracervical tissues between contiguous finger surfaces, has established merit; and we endorse the opinion of those who claim that palpation of the cervical broad ligaments is most satisfactorily accomplished by this maneuver.

Speculum examination is best accomplished with the patient in the dorsosacral (lithotomy) position and with the field well illuminated. Visualization of the source and nature of a discharge is quite important. If excessive, the exudate should be removed gently with gauze or cotton pledgets. The physical characteristics of a cervical cancer are dependent upon the duration and variety of growth as well as upon its point of origin. When the neoplasm is still within the confines of the cervical canal and not visible, or when it is not sufficiently advanced distinctly to alter the appearance

inverting types of cancer, without visible ulceration, the most important findings may be retraction of the cervix with puckering about the external os. When ulceration is present, although chance favors a diagnosis of malignancy, the conclusion must not be unremitting. Sarcoma, tuberculosis and syphilis, although comparatively rare, may produce ulceration of the cervix, as may other nonspecific and benign lesions.[14]

At this juncture of the examination under direct vision, a long, blunt, beaded-point probe should be pressed gently against the visible lesion. If the probe readily penetrates a soft, friable tissue and thus easily induces bleeding, the examiner may be reasonably certain that the area is malignant (Krobach).

Colposcopic examination of the cervix is made with a suitably mounted, low-power

binocular loupe which provides a magnification of ten to fifteen diameters. Its purpose is to reveal small deviations from

is believed specific for the absence of carcinoma.[18]

The Schiller test is a negative diagnostic

1. 50–75 mg. radium element in ametal rubber tandem
2. Vaginal fornix
3. 10 mg. radium element

4. Broad ligament
5. Ametal rubber colpostat
6. Platinum capsule
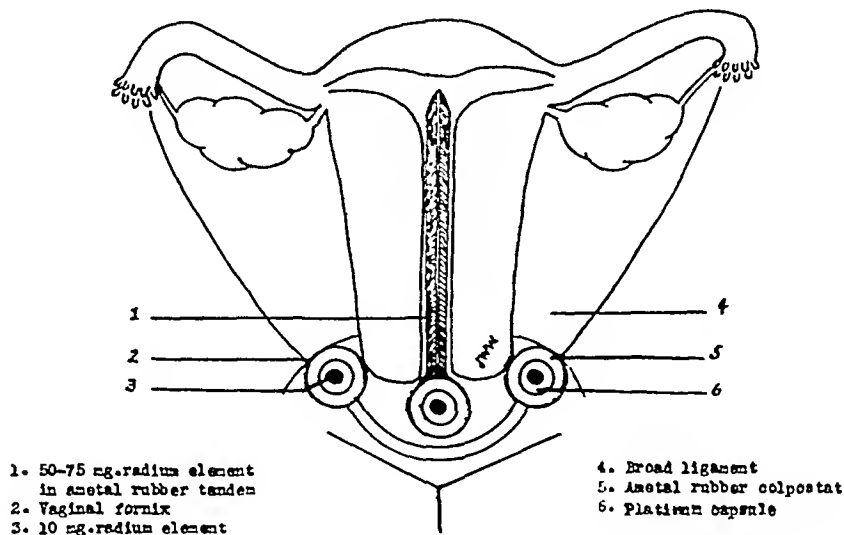
COLPOSTAT

Fig. 8. Diagrammatic transverse view of radium application. 1, 50–75 mg. radium element in ametal rubber tandem. 2, vaginal fornix. 3, 10 mg. radium element. 4, broad ligament. 5, ametal rubber colpostat. 6, platinum capsule.

the normal which may escape unaided vision. This procedure encourages a more minute inspection of the cervix; but a characteristic malignant lesion observed through the colposcope should be detectable without the aid of magnification.

The iodine reaction of Schiller "is of value in locating new growth as long as it is in the stage of a carcinomatous layer." The theory of the procedure is that normal vaginal and cervical epithelium contains glycogen which reacts with iodine to produce a deep brown color. Areas of stratified epithelium that deviate from the normal, such as carcinoma, hyperkeratosis and superficial abrasions, lose their glycogen. In consequence, they remain unstained or assume a faint yellow tint.

The test is readily performed. The cervix is exposed and wiped free of secretion. Gram's iodine solution (iodine 1; potassium iodide 2; water 300) is applied generously and the cervix is examined one minute later. Unstained or slightly tinted areas are considered positive. A negative test

method; it directs attention to a lesion that may or may not be malignant. It is not specific for cancer,[35] and the tissue which does not stain mahogany brown, representing a positive Schiller test, must be submitted to histologic study.

We regard both colposcopic examination and the iodine reaction as of academic interest.

Microscopic examination of removed tissue is the only means by which carcinoma of the cervix may be diagnosed with any degree of incontestible precision. It is imperative in every case that an excised portion or curettings of the cervix be submitted to histologic study. Treatment for a supposed carcinoma unsubstantiated by microscopic verification is likely to deal great harm to the patient, cast unwarranted aspersions upon an otherwise highly valued form of therapy and simultaneously result in the collection of false statistics.

In procuring biopsy material it is best to use some form of thermocautery knife in order to prevent the diffusion of cancer

cells and bacteria and to control hemorrhage. It is inadvisable to remove sections from unanesthetized patients because of

1. Malignant cervix
2. Platinum capsule
3. Radium element
4. Ametal rubber colpostat

COLPOSTAT

FIG. 9. Diagrammatic posterocoronal view of colpostat application. 1, malignant cervix. 2, platinum capsule. 3, radium element. 4, ametal rubber colpostat.

the hesitancy of the operator to induce pain in the removal of sufficient tissue.

Sections should be adequate and include not only tissue that grossly appears to be malignant but also a portion beyond the margin of growth, preferably a full thickness of the cervix, in order to show characteristic features to the best advantage. Unless removed tissues contain some of the cancer cells, when present, the diagnosis is certain to be erroneous, and a life may be sacrificed. In a carcinomatous suspect, with no visible lesion on the vaginal portion of the cervix, a curettement is advised. It is believed that little hazard is involved in properly excising material from a cancer; and we persist in advocating biopsies regularly in the clinic.

Cystoscopic examination is indicated in manifest or suspected involvement of the anterior vaginal wall, and when there are bladder symptoms. Bullous edema or distinct carcinomatous invasion of the bladder mucosa are complications that inalterably modify the plan of treatment. Cystoscopic findings may be called into medicolegal evidence for the purpose of determining whether a subsequently developed fistula is the result of the cancer or of the therapy.

PROPHYLAXIS

It is generally agreed that carcinoma of the cervix tends to arise from an area of

chronic inflammation. Cervical lacerations inevitably become inflamed and are, therefore, to be regarded as potential precursors
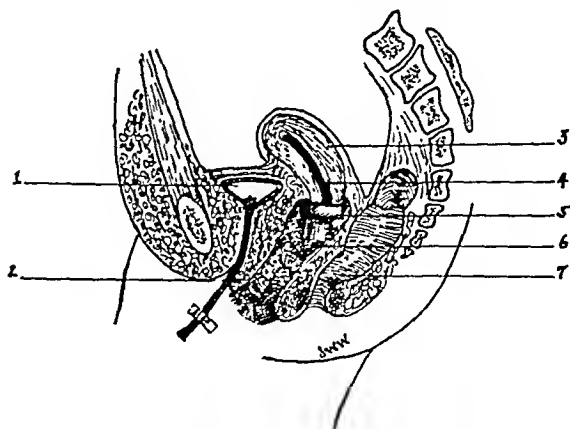
FIG. 10. Diagrammatic sagittal section showing radium application. 1, empty bladder. 2, retention catheter. 3, uterine corpus. 4, radium tandem. 5, cell of colpostat. 6, gauze vaginal pack. 7, rectum.

of cancer. But it must be remembered that chronic inflammatory disease of the cervix frequently appears in the absence of birth injuries. Erosions are ultimately and therefore universally inflammatory whether they arise through a congenital failure of the columnar epithelium to retract within the cervical canal or whether they originate through mechanical or chemical irritation as is frequently the case in the nulliparous.

Statistical reports indicate that cancer develops in the cervical stump after supravaginal hysterectomy in 1.8 to 2.0 per cent of cases.[33] Béclere[5] found the incidence to be 2.6 per cent. However, it is an experienced fact, corroborated by the impression of others,[31,21] that in many of these cases the cancer is residual and was present before the operation. Generally considered, however, a greater number of patients will die from a complete operation (panhysterectomy) than from cancer arising in a cervical stump; and, therefore, the difference in mortality potential between subtotal and total hysterectomy precludes the admonition to perform panhysterectomies in all cases.

Mindful of the considered sources of cervical carcinoma origin and of the advanced stage of the disease when pa-

tients are referred for treatment, it is evident that prophylactic measures properly directed are of the greatest importance
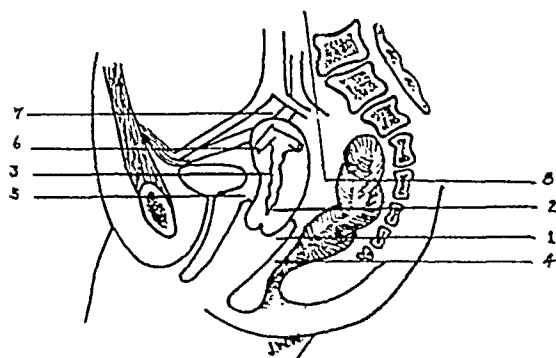
FIG. 11. Sagittal section of regional cancer metastases. 1, vagina. 2, supravaginal cervix. 3, broad ligament. 4, rectovaginal septum. 5, vesicovaginal septum. 6, round ligament to inguinal nodes. 7, hypogastric nodes. 8, lumbar nodes.

in the prevention and control of cancer. To that end it is recommended that:

1. Medical schools and general hospitals establish and maintain tumor clinics primarily for their greater benefit to the patient, but also for the specific and additional purpose of teaching the essentials of examination, diagnosis, prophylaxis and treatment of cancer as it affects various structures.

2. Tumor clinics, national, state and county medical societies sponsor and arrange educational programs not only for the purpose of providing professional postgraduate opportunities but as well for the purpose of disseminating informative cancer facts to the reading and non-reading public.

3. Abdominal examinations of women past the age of puberty include inspection of the cervix.

4. All women after the age of puberty have a complete gynecologic examination yearly.

5. The usual examination at the end of the puerperium be supplemented by similar examinations six months and one year later.

6. Cervical birth injuries be repaired as soon as discovered.

7. Erosions, cervicitis, polyps, leucoplakia and infectious processes, whether in parous or nulliparous women be treated promptly by surgery or cautery until completely replaced by normal epithelium.
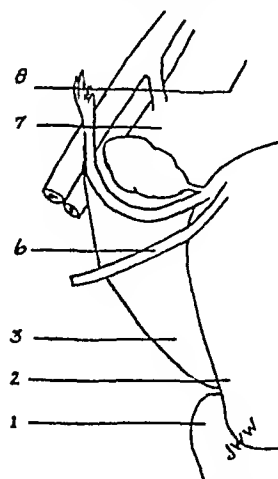
FIG. 12. Coronal section of regional cancer metastases. 1, vagina. 2, supravaginal cervix. 3, broad ligament. 6, round ligament to inguinal nodes. 7, hypogastric nodes. 8, lumbar nodes.

8. When certain malignancy is not already present, the mucosa of cervical stumps, including that of the endocervix, be destroyed by cautery within four weeks after subtotal hysterectomy.

TREATMENT

Dependent upon the degree of progression of the disease, as ascertained from its anatomic distribution or clinical stage, there are three acceptable forms of therapy for carcinoma of the cervix, namely, radical surgery, irradiation plus surgery, and irradiation. In general, however, irradiation efficiently administered is the safest and most certainly curative method of treatment.[4]

The limitations of surgery are well recognized, and there is a growing tendency to restrict operation to the earliest stage of the disease where risks of immediate death are minimal and where the outlook for cure is good. "In the majority of cases, however, the carcinoma has passed beyond the stage of operation when the patient is first seen, and even those in the operable group are

chiefly in the later rather than the earlier stages of the disease. This unfortunate fact is universally accepted both in Europe and America."[7]

In spite of the circumstance that only 10 or 15 per cent of patients are seen sufficiently early to offer any operative prospect of cure,[24] palliative hysterectomy is necessary in those instances where radiation facilities are inadequate, where there is a complicating, persistent pyometra, and where the unenlightened patient refuses radiation therapy. Except in those comparatively few situations wherein the surgeon has no alternative but to invoke palliative measures, certain conditions demand thoughtful consideration before operation is undertaken. The parametria and the regional lymph nodes must be free of cancer; the bladder and the rectum must not be involved; the vagina, if invaded, must permit of resection that will allow a margin of tissue at least 3 cm. from the growth; neither the ureters nor the kidneys should show pressure damage, hydroureter or hydronephrosis, and distant visceral metastasis must be absent. Above all else, the patient must be a good operative risk.

Once the surgical method of approach is decided upon, it should be fully realized that carcinomatous cells that have invaded neighboring pelvic structures may ultimately terminate the life of the patient unless the operation is distinctly radical. To combat the possibility of recurrence from residual cancerous tissue, without the aid of irradiation, entails isolation of the ureters, a wide parametrial dissection and complete removal of the uterus together with a broad cuff of adjacent vaginal tissue. This can be accomplished best through an abdominal approach and by strict adherence to the technique of Wertheim.

Following the advent of radiation therapy there developed a progressive inclination to confine the operative treatment of carcinoma of the cervix to very early cases or to those cases which for one reason or another, as above specified, could not be satisfactorily treated by any other method. The reasons for this trend are apparent. Aside from the technical difficulties, radical hysterectomy entails a high morbidity and mortality rate; and any procedure that has an established mortality rate varying from 16 to 20 per cent[36,37] is not to be undertaken complacently. Besides, "Its [the operation's] execution is so difficult as to render it highly hazardous except in the hands of a very few specialists."[36] Likewise, it is often problematic whether or not distant invasion has occurred before operation; and it is an established fact that patients who have advanced beyond the operable stage, without exception, are doomed to suffering and death unless given the benefit of radiation.

As an alternative it is proposed that surgery and radiation be combined. Advocates of this plan recommend that a full course of x-ray and radium be given six weeks before operation in those cases where surgery is contemplated.[32,33] Some gynecologists implant radium at the time of operation while others employ x-ray therapy postoperatively. There seems to be no controversy as to whether surgery should be fortified by radiation but rather a disagreement as to whether radiation should supplant surgery in all cases. Döderlein[15] aptly expressed himself to the effect that radiologic therapy does everything that radical surgery can do, and that it is less drastic and less dangerous.[20]

Primary radium irradiation is broadly applicable; it is productive of numerous cures even in advanced cases; its application carries little morbidity and practically no mortality; and, when not curative, it prolongs life and ameliorates suffering. Perhaps in no other condition is radium more profoundly and incontestably efficacious than in the treatment of carcinoma of the cervix. Judiciously applied in adequate dosage, its effects are frequently startling.
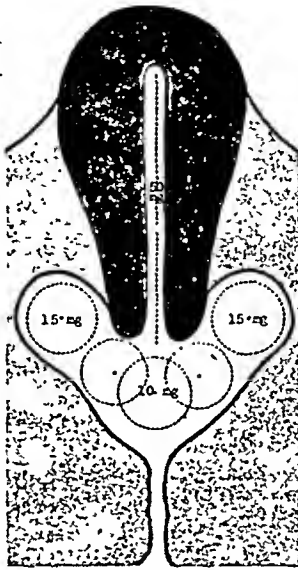
In the regulation of radium dosage there are three fundamental factors that must be taken into account, namely, the qualita-

tive factor, the quantitative factor and the time factor. The qualitative factor indicates the form of radiation, radon or

ence, to become somewhat expert in its application.

For some undetermined reason the

FIG. 13. Plan of recording radium application.

radium element. The quantitative factor, expressed in Grams (Curies) or fractions thereof, has to do with the amount of irradiation, the distance from its source and the character of screening, lead, brass, gold, silver, platinum, rubber. The time factor indicates the period of exposure; and it is of the greatest importance in the treatment of human cancer.[25,40] These factors are constant the world over; yet, there are scarcely any two prominent clinics that employ identical radiation technique, although the statistics of those with an experienced personnel using adequate dosage show approximate results.

Equally important as the understanding of what comprises an adequate dose of radium in a given case is a reasonable experience in its application. Radium is by no means a simple remedy that may be applied casually without serious consequences. Because of its potential dangers it should be used only by those who have had an opportunity, through supervised experi-

impression is extant that a large quantity of radium is necessary for the proper treatment of cancer; and a number of inadequately informed physicians are of the opinion that "there is insufficient radium, except in a few of the larger centers, to do more than stimulate neoplastic growth." This notion is distantly remote from indisputable fact. Basing his statement upon his own experience with autopsy material, Ewing appropriately says:

"The main practical conclusion I wish to draw from all these data is that the danger of stimulating the growth of tumors by the present methods of radiotherapy is extremely remote, while the idea that inadequate radiotherapy can endow the cells with increased vitality is quite unreasonable."[16]

Nevertheless the employment of inadequate quantities of radium is not to be condoned; and the therapist with no more than 50 mg. available is to be considered a menace to cervical cancer patients. In-

sufficient dosage which cannot adequately sterilize the neoplasm renders it radio-resistant and makes future, safe irradiation dosages difficult to compute.

Inasmuch as few cases are anatomically identical and each, therefore, requires individualization of therapy, varied in accordance with the character, location and extent of the disease, and its complications, no single method of radium application is universally applicable; and the fallacy of an endeavor to outline a routine method of applying radium in the average case is clearly obvious.

However, it may be unequivocally stated that the minimum total dose of radium should not be less than 2400 mg. hours and that the maximum total dose should not exceed 8000 mg. hours. The radium should be distributed in such manner as to irradiate the entire cervix, the uterus and the parametria through intrauterine and vaginal applicators. It is our custom to use for this purpose an intrauterine tandem of 50 to 75 mg. of radium element filtered through 1 mm. of platinum-iridium and 3 mm. of ametal rubber. This intrauterine applicator is permitted to remain in situ until one-third to one-half the total combined dosage is delivered. Concomitantly placed with the intrauterine applicator are the vaginal applicators. These are applied to the cervix and the vaginal fornices by means of a colpostat or similar device, and consist of 0.5 mm. platinum filtered needles or capsules of not less than 10 mg. nor more than 20 mg. in each ametal rubber cork of the colpostat, which rubber corks add another centimeter of filtration. The irradiation period continues for 120 consecutive hours. The applicators are removed every twenty-four hours and before their reinsertion the vagina is gently douched with a liter of warm sterile water. The bladder and rectum are protected by carefully forcing them away from the radium containers with intravaginal plain gauze packing. A retention catheter is maintained in the bladder during the course of primary treatment.

Unless complications ensue, such as inflammatory adnexal changes or a rise in fever above 101°F., the treatment is massive and continuous in accordance with the French method, because it is believed that by this means the entire tumor is subjected to a thoroughly uniform dosage and that there is delivered a relatively and absolutely higher dose of devitalizing rays, with a consequent more beneficial parometric effect,[22] without risk of severe reaction or danger to the patient. Also it is understood that biologic tissues have the ability to recover rather promptly from irradiation and, therefore, a more prolonged effect may be induced by continuous exposure. Furthermore, if the dosage is administered in three or more stages the danger of ascending infection is said to be increased.[17] Short period exposures definitely enhance the factor of uncertainty.

If given before the radium application, deep Roentgen therapy may at the earliest moment effect several actual as well as theoretical advantages: it affects malignant cells beyond limits attainable by radium, it reduces the activity of cell proliferation, it decreases the size of the larger primary tumor, it controls hemorrhage and it unfavorably influences local infection. By these means x-ray commonly facilitates the application of radium. Pre-radium Roentgen therapy probably will never supplant post-radium Roentgen therapy; but whether the former will be utilized to supplement the latter in all cases remains to be determined, and will depend upon results not yet evaluated. It has been the plan, except for the cases classified as belonging in group 1, immediately the primary radium treatment is completed and the patient's condition permits, to institute high voltage Roentgen therapy. It is by means of this adjunct that the development and spread of cancer cells not previously destroyed may be prevented. Patients who have very extensive tumor masses and where an endeavor is made to reduce the neoplasm to facilitate the application of radium, especially into the

cervical canal, are given a primary course of deep Roentgen therapy. The latter modality is also employed for palliation and the relief of pain in advanced cases. High voltage x-rays are more universally available than are radium packs or bombs and they are equally effective in the treatment of carcinoma of the cervix.

Sometimes very far advanced cases show phenomenal regression before the usual intensive treatment is completed. In others a second modified course of irradiation, about eight to twelve weeks after the first, is necessary to produce desired effects. Tissues in a state of highly active cellular change are most malignant and, conversely, extremely responsive to radium therapy. However, varying degrees of cellular differentiation may be observed in different areas of the same cancer. Classified as to the degree of malignancy according to Broder's standards, carcinoma of the cervix is of grade 3 or 4 in from 75 to 85 per cent of cases.[26] A great majority of these are radiation sensitive.

While it is true that attempts have been made to apply radium intra-abdominally along regions of cancer spread, the problem presented, that of protecting the surrounding viscera, particularly the bladder and rectum, remains unsolved. It is clearly evident that in order to increase the curability rate of later cervical cancer cases some change in present methods of treatment is necessary. In the light of current knowledge, and lacking a specifically destructive dose of radium or Roentgen rays for cancer cells, applicators must be devised in such manner as to emit rays effective against cancer which applicators will at the same time afford protection to neighboring viscera. A device of that kind would be ingenious, indeed, and its principle of effectiveness would depend upon the direction of devitalizing rays between adequate filters. Added to the latter difficulty is the uncertainty as to whether or not a plan of treatment necessitating surgical exploration of the pelvic organs will prove sufficiently advantageous to offset the accruant disadvantages, increased morbidity and mortality, by a reasonable increase in the curability rate.

CURABILITY

The absolute curability rate for cancer of the cervix throughout the world varies between 20 and 30 per cent.[29]

In a report of a previous study of eighty-nine cases[39] it was shown that approximately 40 per cent died within the first year after radium treatment. Sixty-one per cent of the total number of patients treated died within three years, and 20 per cent lived five or more years. Less than 6 per cent were alive and clinically cancer-free after more than ten years. It was observed that those who survived the five year period had a better chance for prolonged survival than similarly relieved breast cancer cases.[38]

CONCLUSIONS

1. Although specifically curative measures have not been devised, there is sufficient knowledge based upon sound working principles to decrease the incidence and to cure a larger percentage of carcinomas of the cervix. The substance of that knowledge must be disseminated repeatedly until comprehended by every physician who deals with cancer.

2. Tumor clinics may ideally serve as lay information and professional teaching centers for which there is admitted need.

3. The early diagnosis of carcinoma of the cervix is dependent upon objective findings, making critical examination imperative in every case.

4. Microscopic examination of adequate biopsy material is the only means by which cancer of the cervix may be diagnosed with any degree of incontestable precision; and little hazard is involved in properly excising biopsy specimens.

5. Colposcopic examination and the Schiller iodine test are of academic interest. Both are greatly subsidiary to unaided visualization and biopsy. The Schiller test is a purely negative diagnostic method.

6. Palpation of the cervical broad ligaments is most satisfactorily accomplished by a combined vaginorectal examination.

7. Cystoscopic findings may be called into medicolegal evidence for the purpose of determining whether a subsequently developed fistula is the result of the disease or of the therapy.

8. Adversity in the prognosis in carcinoma of the cervix is directly proportionate to the progression of the disease from one clinical stage to another; and the prospect of curability is only further unfavorably altered by complicating factors, providing the treatment is adequate and judiciously administered.

9. The rapidity with which a cancer spreads and metastasizes is a fairly constant index of the degree of malignancy of the primary tumor, but it is not an index of its curability at a given period. The majority of cervical cancers are radiosensitive; and the more active and anaplastic the cells the less their radioresistance.

10. Often nodes that are nearest the primary tumor, and which are naturally expected to be the first involved, are found absolutely cancer-free, while a more distant group will show marked secondary deposits, a paradox in metastasis.

11. It would seem that there is a direct relationship between lymph stasis or lymphatic obstruction and the localization of secondary transplants.

12. Cancer appearing in the cervical stump within one year after supravaginal hysterectomy is residual and was present before the operation. If it appears after a longer interval, it must be considered a subsequent, primary new growth. As prophylaxis against the latter event, the mucosa of the cervical stump including the endocervix, if not destroyed by cautery at the time of operation, must be so destroyed within four weeks thereafter.

13. Radium irradiation efficiently administered is the safest and most certainly curative method of treatment in carcinoma of the cervix, but it must be supplemented by high voltage Roentgen rays in all except the earliest cases. Primary Roentgen therapy, supplemented by post-radium Roentgen irradiation, offers advantages that require further evaluation.

14. Radium is by no means a simple remedy that may be applied casually without disastrous effects or serious consequences. Therefore, it should be used by those only who have had an opportunity through supervised experience to become somewhat expert in its application.

15. Although the absolute curability rate for cancer of the cervix is between 20 and 30 per cent, one-half the total number of patients treated will die of the disease within three years.

REFERENCES

1. ADAIR, F. L. Symptomatology of cancer of the uterine cervix. S. Clin. North America, 16: 3–12 (Feb.) 1936.
2. ANSPACH, B. M. Gynecology, 2nd Ed., pp. 331–342. Philadelphia, 1932. J. B. Lippincott.
3. AUER, E. S. Carcinoma of the cervix uteri. J. A. M. A., 98: 2259–2262 (June 25) 1932.
4. BAER, J. L. Carcinoma of the cervix complicated by pregnancy. S. Clin. North America, 16: 51–61 (Feb.) 1936.
5. BÉCLÈRE, C. Cancer of the stump after partial hysterectomy. Bull. Soc. obst. et gynéc. de Paris, 24: 372–378 (June) 1935.
6. BLAND, P. B. Quoted by Pfahler, G. F.[30]
7. BLOODGOOD, J. C. Carcinoma of the cervix. Am. J. Cancer, 16: 1238 (Sept.) 1932.
8. BOYD, W. Surgical Pathology, 3rd Ed., pp. 484–485. Philadelphia, 1933. W. B. Saunders.
9. BRODERS, A. C. Carcinoma in situ contrasted with benign penetrating epithelium. J. A. M. A., 99: 670–671 (Nov. 12) 1932.
10. BURNAM, C. F. Radium in Gynecology. Practice of Surgery, 11: 20–23. Hagerstown, Md., 1932. W. F. Prior Co.
11. CHARLTON, A. R. Cancer Control. Chicago, 1937. Surg. Publ. Co.
12. CRAMER, W. The prevention of cancer. Lancet, Jan. 6, 1934.
13. CROSSEN, H. S., and NEWELL, Q. U. Five year cures of carcinoma of the cervix uteri. Surg., Gynec. & Obst., 58: 450–451 (Feb.) 1934.
14. DAVIS, C. H. Lesions of the cervix uteri—diagnosis and treatment. New England J. Med., 213: 699–705 (Oct. 10) 1935.
15. DÖEDERLEIN. Quoted by Gardner, G. H., and Finola, G. C.[20]
16. EWING, J. An analysis of radiation therapy in cancer. Mutter Lecture, Radium Report of the Memorial Hospital, Series 2: 243–288. New York, 1924. Paul B. Hoeber.
17. Foreign Letter, Berlin. J. A. M. A., 107: 1482 (Oct. 31) 1936.

18. Frank L. Carcinoma of the cervix uteri. *Am. J. Surg.*, 23: 413–418 (March) 1934.

19. Friedman, N. Precancer of the cervix uteri. *Surg., Gynec. & Obst.*, 58: 717–726, 1934.

20. Gardner, G. H., and Finola, G. C. Recent literature on malignant tumors of the uterus, cancer of the cervix. *Surg., Gynec. & Obst.*, 58: 1–23, (Jan.) 1934.

21. Goldstein, M. T. Diagnosis of carcinoma of the cervix. *S. Clin. North America*, 16: 13–17 (Feb.) 1936.

22. Hamann, A., and Goebel, A. Complications of radium therapy in gynecologic carcinoma. *Zentralbl. f. Gynäk.*, 59: 677–688 (Mar. 23) 1935.

23. Handley, W. S. Prevention of cancer. *Lancet*, 230: 987–991 (May 2) 1936.

24. Healey, W. P. Treatment of carcinoma of the cervix. *Bull. Am. Soc. for Control of Cancer*, 16: 5–7 (March) 1934.

25. Holtheisen. Quoted by Wood, F. C.[40]

26. Judd, E. S., and Phillips, J. R. Curability of carcinoma of the cervix. *Proc. Staff Meet. Mayo Clin.*, 8: 789 (Dec. 27) 1933.

27. Martzloff, K. H. Diseases of the Cervix Uteri, Practice of Surgery, vol. 10, Chapt. 14, pp. 1–213. Hagerstown, Md., 1932. W. F. Prior Co.

28. Novak, E. Pseudomalignant and precancerous lesions of the cervix. *J. A. M. A.*, 108: 1145–1151 (April 3) 1937.

29. Newell, Q. U. Five year end results in the treatment of cancer of the uterine cervix at the Barnes Hospital, St. Louis. *Am. J. Obst. & Gynec.*, 24: 167–169, 1932.

30. Pfahler, G. F. Can we prevent cancer? *Bull. Dept. Pub. Health*, Phila., Jan.-Feb., 1932.

31. Phaneuf, L. E. Carcinoma of the cervical stump. *Am. J. Surg.*, 29: 479–480 (Sept.) 1935.

32. Report of Cancer Com. of California M. A. *Bull. Am. Soc. Control of Cancer*, 16: 1–5 (April) 1934.

33. Report of Wash. State Med. Soc. on Gyn. Tumors. *Bull. Am. Soc. Control of Cancer*, 16: 4–6 (Feb.) 1934.

34. Scheffey, L. C., and Crawford, B. L. Adenocarcinoma of the cervix in a twenty-two month old child. *Am. J. Obst. & Gynec.* 24: 118–120, 1932.

35. Schiller, W. Diagnosis of carcinoma of the cervix in a very early stage. *Lancet*, 230; 1228–1232 (May 30) 1936.

36. Schmitz, H. E. Form of treatment in cancer of the cervix. *Surg. Clin. North America*, 16: 19–29 (Feb.) 1936.

37. Shaw, W. F. Radium versus Wertheim's hysterectomy in the treatment of carcinoma of the cervix. *Surg., Gynec. & Obst.*, 64: 332–337 (Feb. 15) 1937.

38. Wainwright, J. M. Cancer of the lip, breast and cervix. *Bull. M. T. H.*, 1: 9–14 (May) 1927.

39. White, J. W. Carcinoma of the uterine cervix. *Med. Rec.*, 141: 83–88 (Jan. 16) 1935.

40. Wood, F. C. Progress in x-ray therapy. Reports of the Second International Congress of Scientific and Social Campaign against Cancer, 1: 320–330, 1936.

EROSION AND INFECTION OF THE ANTEPARTUM CERVIX AND THEIR TREATMENT BY ELECTROCOAGULATION

INGLIS F. FROST, M.D., F.A.C.S.

NEW YORK CITY

RADICAL treatment of the cervix uteri during the antepartum period has not been a common procedure among obstetricians. Many observers feel that this practice is not only unsafe but dangerous as it may be the means of interrupting an existing pregnancy.

However, the last decade has witnessed many improvements in the care of the gravid patient, not only in the actual technique of the delivery itself, but also in a more exacting care of the patient, during the antenatal and postnatal periods. One of the most noticeable improvements has been in the follow-up treatment of the postpartum cervix; obstetricians, in general, have become cervix-conscious. Practioners now are not content to discharge their patients until the cervix is thoroughly healed and has assumed its normal condition, that is, without erosions or infections.

Treatment of the antepartum cervix has suffered in comparison with that of the postpartum cervix. Many obstetricians are content, no matter how extensive the erosion or infection of the cervix may be, to suggest by way of treatment, the application of some innocuous chemical and the use of the time-honored douche. In most foreign countries the antepartum cervix is rarely honored with a visual examination, while the postpartum cervix in many places is as yet a stranger to the advantages offered by electrocoagulation.

In a questionnaire sent to several hundred obstetricians throughout this and other countries, an effort was made to ascertain the frequency of occurrence of erosions in the antepartum cervix. The returned figures showed an amazingly large percentage, varying from as low as 20 to as high as 80 per cent.

The question of the reason for the prevalence of this condition naturally presents itself; and there is the further question as to why it should continue to be so. The answer is twofold, first because of the anatomic position of the cervix and second its susceptibility to infection.

The location of the cervix in the vagina exposes it to the continual risk of infection and during the period of labor it is subject to a traumatism that few organs of the body undergo. Being a highly glandular structure, and containing as it does many racemose glands, the cervix is prone to infection. Unless this infection is promptly treated and cured, there soon develops a chronic endocervicitis that rarely, if ever, heals spontaneously. Maryan has definitely shown in the cervices examined by him, that the enterococcus was removed from the depths of the racemose glands in 80 per cent of the cases. It is thus understandable why such conditions as salpingitis, metritis, perimetritis, pyelonephritis, and pelvic peritonitis may develop and why the cervix may be, and is such a potent focus of infection.

Medical men have laid great stress upon the fact that both the tonsils and teeth are important sources of infection and insist upon their removal where indicated. The cervix as a focus has not received the same amount of careful attention, and its treatment in many cases has been restricted to mere swabbing of some impotent chemical or the vaginal douching of an objectional leucorrheal discharge.

Hundreds of women, each year, approach delivery with a markedly infected cervix, and yet many obstetricians are loathe to institute radical treatment beforehand because of the fear of interrupting pregnancy. However, would it be unreason-

able to question the relationship of an infected antepartum cervix to puerperal infection? Certainly the bacteria harbored

FIG. 1. Erosion with eversion and polypoid formation following extensive bilateral tear.

in the cervix put an additional onus on the mother approaching delivery. Because of their fear of causing abortion, most obstetricians have contented themselves with treating only the postpartum cervix.

FIG. 3. Eversion reduced and cervix healed. Time of healing about six weeks following electrocoagulation.

While this is an excellent procedure, it does not protect the expectant mother. It is only reasonable to assume that a normal, non-cicatrized, non-eroded and non-infected cervix will efface with more

ease and will show less tendency to tear than a diseased and chronically infected cervix will.

FIG. 2. Erosion with eversion treated by electrocoagulation. Note white coagulation.

Because of the excellent results obtained by electrocoagulation, in the postpartum cervix, it was decided to try this method on the antepartum cervix, with results which have been more than gratify-

FIG. 4. Erosion of cervix of three years' duration.

ing. In a series of thirty cases there has not been a miscarriage. Treatment has been instituted anywhere from the sixth week to the fifth month of pregnancy. At no time was any attempt made to coagulate the cervical canal, all treatments

being confined to the surface erosions or infections.

All the patients were examined and
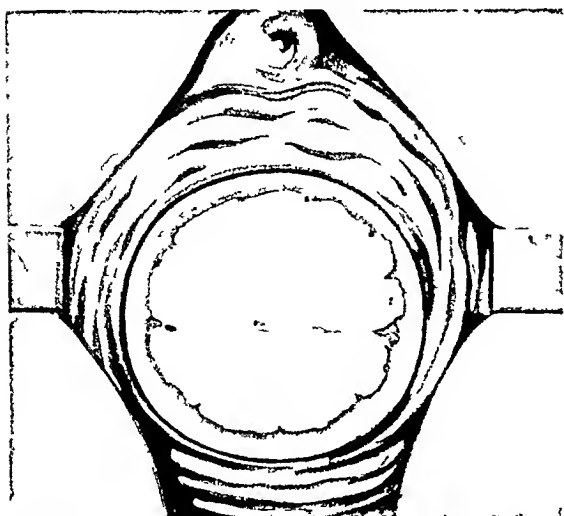
FIG. 5. Coagulation of cervix erosion. White coagulation.

treated privately. They supposedly had been in the care of private physicians previous to their pregnancies and following former deliveries. A tabulation of these

For practical and descriptive purposes the erosions are divided into first, second, third and fourth degree, depending on their size. For example, a first degree erosion would be about the size of a ten cent piece, a second degree about the size of a five cent piece, a third degree about the size of a quarter, and a fourth degree anything over this. The majority of the cases were primiparae and the major number of fourth degree erosions occurred in them. Twenty-two of the thirty women were primiparae and twelve of these had first degree erosions, two second degree erosions, and eight fourth degree erosions.

Four of the patients had had one previous child. Two of these had first degree erosions, and two fourth degree erosions, one of which had a bilateral tear. Two women had had three children. One of these patients had a first degree erosion and the other a second degree erosion with a left lateral tear. The remaining two women had fourth degree erosions with bilateral cervical tears, and large cystoceles
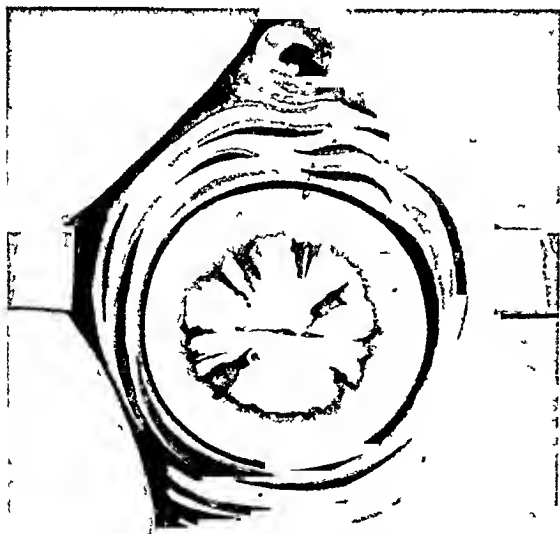
FIG. 6 Two weeks after coagulation Areas of new epithelium appearing and slough separating.

FIG 7 Healed cervix four weeks after coagulation

cases was carefully kept for the purpose of recording the progress of healing during the antenatal period and also to compare the amount of damage and erosion of the postpartum cervix with that of the treated and healed antepartum cervix.

and rectoceles. Both of these patients had infected cervices with profuse discharge. One woman had had four children and the other five.

Treatment in each case was by electrocoagulation. This was confined to the

portio only and the lightest possible contact was made, producing a mild searing or white coagulation. The time of healing varied from four to eight weeks, the major number of cases healing in about four weeks. Fourteen healed in four weeks, four in five weeks, ten in six weeks, and two in eight weeks.

The time of labor was not affected in any degree. This was especially true of the primiparas who had the larger erosions and required the greatest amount of coagulation. The average time of these deliveries was from eight to ten hours. One multipara, who was a para three, was in labor for ten hours, due to a hard cicatrized cervix from former labors.

The patients were all examined from four to six weeks after delivery, and only in a very few cases was there a demonstrable tear unless such a tear had existed previously. Even in these cases the cervix healed very rapidly and showed a minimum amount of erosion.

All of the primiparae were delivered, where possible, by prophylactic forceps. There were no breeches or versions, but in three instances rotation was done manually from posterior position.

Twelve of the patients, on postpartum examinations, showed but slight erosions and fourteen had first degree or slightly greater erosions. These were promptly coagulated and all were healed in from four to five weeks' time.

A follow-up record at six months and a year have shown that twenty have remained healed, while the remaining ten, which have not as yet passed the six months mark, have remained healed so far.

CONCLUSIONS

It would seem that antenatal examination of the cervix is an important routine measure in the prenatal care of the expectant mother, and that electrocoagulation, if confined to the portio only, and practiced with infinite caution as to white coagulation only, is a safe and necessary technique in the care of the maternity patient.

Routine examination of the prenatal cervix is a necessary factor in antenatal care. Erosions of all degrees may be expected in the primiparae as well as in the multiparae. These erosions may be safely treated by electrocoagulation in the prenatal patient, at any time between the second and the fifth month, but preferably in the second month. Healing takes place in about four weeks and all annoying discharges cease.

Postpartum erosions are rarely present or, if so, are rarely more than first degree erosions. These remain healed after a six months period and the cervix shows no scar tissue.

REFERENCES

Frost, I. F. Cervical infections and treatment by electrocoagulation. *Am. J. Surg.*, 3: 221–226, 1927.

Frost, I. F. The correct technique in electrocoagulation of the cervix and its attending dangers. *J. M. Soc. New Jersey*, 34: 621, 1937.

Boland, B. F. Ascent of infection from the uterine cervix to the urinary tract. Its treatment by various electrical methods. *M. Rec.*, 145: 199 (March 3) 1937.

THE DISCISSION OPERATION

NED SHNAYERSON, M.D.

NEW YORK CITY

THE operation suggested in this paper is for a common clinical entity frequently encountered in practice,

odor. An inflammatory reaction is set up within the cervix and an endocervicitis is produced. This then becomes a vicious
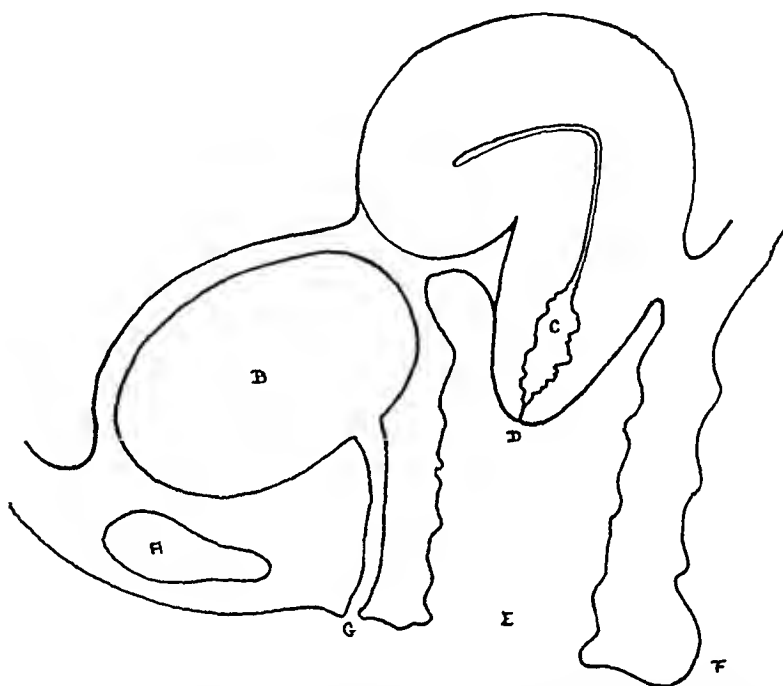
FIG. 1. Anatomy and lesion described. A, pubic bone, B, bladder. C, distended cervical canal. D, pinpoint os. E, vagnia. F, rectum. G, urethra.

namely that of the long conical cervix with a pinpoint os. It is a congenital maldevelopment and is usually associated with an acutely anteflexed uterus.

Relatively serious subjective and objective symptoms (sterility and dysmenorrhea) frequently arise from this maldevelopment, basically due to an obstructive mechanism. The pinpoint os is the principal factor in this obstructive mechanism. Due to the minute exit at the cervix afforded to the cervical secretions, there is a damming up of its contents. The accumulation of this material results in a distended canal (Fig. 1) containing detritus which becomes infected and has a foul

cycle; the end products of the endocervicitis being accumulated and these in turn further injuring the endocervix and cervix.

In Sturmdorf's work on sterility he has clearly shown the far-reaching effect of an infected cervix: how sterility may be produced and how understandable is the uterine lymphangitis producing dysmenorrhea.

"In the cervix, as elsewhere, every infection incites the greatest reaction in the lymphatic elements. The cervical lymphatics may be traced from their lacunar origin in the cervical canal, through minute funnel-shaped ostia, directly to the mus-

cular coat, where they expand into an extensive capillary net which enmeshes every fascicle and bundle of the uterine
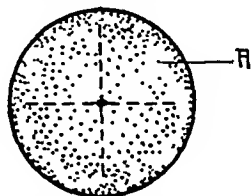
FIG. 2. Vaginal surface of the cervix. Pinpoint os at center with crucial incision (indicated by the two broken lines) intersecting at the os. The four quadrants to be removed are thus outlined.

musculature to the peritoneal surface, whence they drain into their main collecting channels at the base and top of the broad ligament (Leopold). Thus the normal course of the lymphatics conveys an infection from the cervix, *not to the corporeal endometrium*, but along the intramuscular planes of the uterus, where it impairs uterine contractions by infiltrating the muscle sheaths, resulting in nutritional deficiency; progressing to the periadnexal network, it inhibits tubal peristalsis and agglutinates the fimbrial ostia by adhesions, finally reaching the ovaries; it impedes the normal rupture of Graafian follicles by thickening of the ovarian tunica albuginea, thus completing the chain of pathologic events that link cervical disease and sterility.

"Crystallized into a concrete postulate, chronic endocervicitis presents the key to the therapeutic problem in sterility of cervical origin, and the success of any curative attempt upon the cervix will be proportionate to its elimination of an existing endocervical infection."

We see then that in the maldeveloped cervix with the pinpoint os, this syndrome is due to a lack of normal and sufficient drainage of the cervix and that we must attempt to establish a sufficient drainage to overcome these undesirable symptoms.

To obviate this developmental anomaly and its sequelae, a number of operations have been devised, plastic in character, but involving considerable alteration in the normal anatomy of the cervix and pro-

ducing functional changes. Simple dilatation is insufficient. In the typically stenosed os of this type, it is extremely difficult to
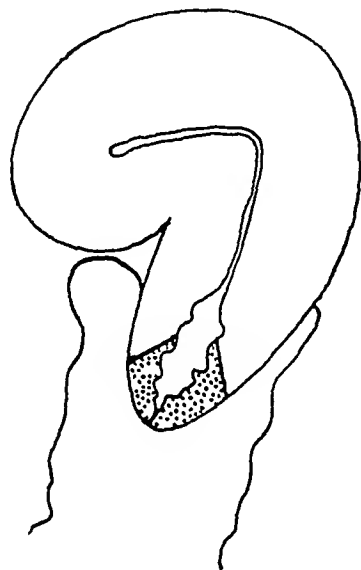
FIG. 3. The plane of dissection from the base of the outlined triangular area on the vaginal surface of the cervix. The shaded area is the portion of the cervix removed, leaving a coned out area in the cervix.

dilate the cervix. Very often a surgical probe cannot enter the os. If dilatation is done, the fibers of the cervix are torn, scar tissue is formed, the os returns to its previously contracted state and the contraction of the fibrous scar tissue further keeps it so.

The operation outlined is very simple and extremely efficacious. It does not distort the normal architecture of the cervix but creates a normal external os. It relieves the obstruction to the flow of the cervical contents and alleviates the dysmenorrhea because the lymphatic infection is relieved by the drainage. When sterility has been associated, we have had a large number of pregnancies follow this relief.

It is due to Dr. L. J. Ladin, who has kept the patina of time from obscuring this operation, that I have used this procedure for the past ten years. I have found it as successful as his enthusiastic pleas have claimed.

TECHNIQUE

The patient is prepared as in any vaginal operation. After iodizing the vagi-
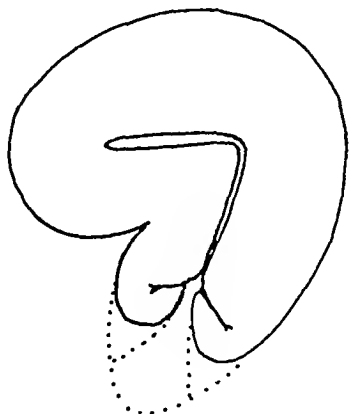
FIG. 4. After removal of the quadrants, the edge of the cervix is infolded and sutured.

nal mucosa and the cervix, the cervix is grasped with a tenaculum forceps and a crucial incision is made into the cervix extending up toward the internal os. (Fig. 2.) The point of intersection of the incisions is at the external os. The four triangular segments thus outlined are each in turn grasped with an Allis forceps and dissected out. The plane of dissection extends from the base of the outlined triangular segment (Fig. 2A) on the vaginal surface of the cervix, inward and upward to the cervical lumen. (Fig. 3.) When these four segments are removed, we have a coned out surface in the body of the cervix, the apex within the cervix and the base at the vaginal surface of the cervix. Dilating instruments can now be

inserted through the cervix with ease. The internal os is *very slowly* dilated so as not to rupture any of the fibers. After
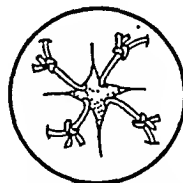
FIG. 5. The edge of the cervix folded in and held in place by a suture in each quadrant.

thorough dilatation, the uterus is curetted, particularly the lower segment about the internal os. The uterus is then dried by *inserting into it a dry uterine gauze strip*. This is followed by insertion of a similar strip soaked in iodine. This strip is then removed and the edge of the cervix may or may not be turned in by a suture in each quadrant. (Figs. 4 and 5.) If the edge is not turned in epithelialization readily takes place. A thin iodoform cigarette drain is inserted into the cervix and passed beyond the internal os. This drain is left in situ for seventy-two hours.

REFERENCES

1. SIMS, J. M. Amputation of the cervix uteri. *J. Med. Soc., New York*, 1861.
2. POZZI, S. Traitement chirurgical d'une cause très fréquente de dysménorrhée et de stérilité. *Bull. Acad. de méd., Paris*, 12: 266–282, 1909.
3. DUDLEY, E. C. A plastic operation designed to straighten the anteflexed uterus. *Am. J. Obst.*, 24: 142–151, 1891.
4. LEOPOLD, C. G. Die Lymphgefässe des normalen, nicht schwarngeren, uterus. *Arch. f. Gynäk.*, 1874, VI.
5. STURMDORF, A. The cervicoplastic treatment of sterility. *Am. J. Obst.*, vol. 76, 1917.

TRANSURETHRAL PROSTATIC RESECTION*

A REVIEW OF 1,200 CASES OF PATIENTS MORE THAN SEVENTY YEARS OLD

GERSHOM J. THOMPSON, M.D.   AND   HAROLD C. HABEIN, M.D.

Section on Urology, The Mayo Clinic   Division of Medicine, The Mayo Clinic

ROCHESTER, MINNESOTA

THE development of the transurethral method of operation for the relief of urinary obstruction caused by prostatic enlargement has been one of the most significant advances in modern medicine. The employment of this method has been the means of reducing the immediate operative mortality rate previously associated with suprapubic and perineal prostatectomy by as much as 90 per cent. Owing to rapid convalescence, the period of hospitalization and therefore its cost have been reduced to considerably less than are required for the performance of an ordinary appendicectomy (in 85 per cent of cases, less than two weeks). Also, because of its widely increased scope, this operation can be performed with a degree of safety that never before has been possible, even among patients of the older age groups, many of whom suffer from advanced cardiovascular renal disease and other degenerative conditions. Equally important, if not more so, is the degree of comfort and happiness which this method of operation affords such patients. As a rule, remarkable improvement in general health and vigor occurs following the relief of urinary obstruction even among patients who have advanced cardiovascular renal disease.

It is well recognized that, in dealing with the enlarged prostate gland by the method of suprapubic prostatectomy, a preliminary suprapubic cystostomy should be performed in a large percentage of cases. Naturally, the poorer the surgical risk encountered, the larger the number of patients that were treated in this manner; furthermore, the more inadequate the renal function and the slower the improvement in general health, the longer such drainage was necessary. Patients of the older age groups who saw their friends subjected to the discomfort and inconvenience of prolonged suprapubic drainage, or who, indeed, did not even see them again after operation, naturally hesitated to accept operation and, hence, postponed

TABLE I

AGE DISTRIBUTION OF 1,200 PATIENTS

Age	Per Cent
70–74	57.6
75–79	30.0
80–84	9.6
85–89	2.5
90–94	0.3

it until a situation had arisen in which there was no other recourse.

In order to evaluate the possibilities of relief of urinary obstruction by transurethral resection, we have reviewed the case histories of 1,200 patients aged 70 years or older (Table I) who were operated on prior to January 1, 1938. In some of these cases it was necessary to perform two and, in a few cases, three operations; hence, in all, a total of 1,361 transurethral resections were performed.

Three hundred of these histories, picked at random, were studied very carefully in order to determine the degree of cardiac, renal and other organic impairment which might be said to have influenced the risk of operation. Table II illustrates the analysis of sample cases chosen at random and indicates the type of risk encountered. These cases have been graded arbitrarily according to operative risk into grades 1, 2, 3 and 4. For instance, it is obvious that a patient more than 70 years of age who has generalized arteriosclerosis, coronary sclerosis and angina pectoris, whose electrocardiogram gives evidence of significant changes, and

who suffers from renal insufficiency, would approach a point at which the risk of operation might be prohibitive. Similarly, in those cases classified as grade 3, the general condition was slightly better, but it will be noted that, in this group of patients suffering from the conditions indicated in the table, the risk of operation is only slightly less than that for those classified as grade 4. It is apparent, therefore, that 56.6 per cent of the cases were extremely poor surgical risks. Thirty-six per cent were classified as grade 2 risks and only 7.4 per cent could be classified as those in which the risk of operation was minimal.

In our opinion, if suprapubic enucleation of the prostate gland were the type of operation employed, those patients who were graded 2, 3, or 4 would have first been subjected to cystostomy and prolonged suprapubic drainage. Many urologists

TABLE II

CONDITIONS WHICH INCREASED OPERATIVE RISK: 300 PATIENTS

Cardiovascular disease		
Generalized arteriosclerosis, grade 3 and 4		
Hypertension, moderate and severe		
Coronary sclerosis, angina pectoris		
Significant electrocardiographic changes	Grade 4	57 patients 19 per cent
Auricular fibrillation		
Previous cardiac decompensation		
Renal insufficiency	Grade 3	113 patients
84 patients, 49.4 per cent of patients		37.6 per cent
who had an operative risk grade 3	Total	56.6 per cent
or 4		
General condition		
Senility, weakness, sclerosis of central nervous system		
Diabetes, prostatic carcinoma, Parkinson's disease		
Recent operations, past cerebral vascular accidents		
Hypertension, obesity, generalized arteriosclerosis, grade 2, mild renal insufficiency		
Generalized arteriosclerosis, moderate hypertension, mild renal insufficiency, diabetes	Grade 2	108 patients 36 per cent
Moderate hypertension, mild to moderate arteriosclerosis, no significant electrocardiographic changes, normal renal function	Grade 1	22 patients 7.3 per cent
Excellent to remarkable condition for age		

TABLE III

DEGREE OF GENERALIZED ARTERIOSCLEROSIS (300 CASES)

Grade	Per Cent
4	7.0
3	42.6
2	45.0
1	5.03

have advocated preliminary cystostomy as a measure which must always precede

prostatectomy for patients more than 70 years of age; hence, even the patients we have classified as grade 1 would have been advised by such urologists to undergo preliminary cystostomy. That this is no longer necessary is indicated by the fact that in only 1.3 per cent of the 1,200 cases which we have reviewed was cystostomy deemed necessary. In recent years as experience has been gained in managing these cases, we have employed cystostomy preliminary to transurethral prostatic resection less and less, and now we believe that the operation is very rarely indicated.

We believe that dispensing with cystostomy, except in rare cases, has been an incentive for very old men in extremely poor condition to consider the possibility of surgical relief. Formerly, owing to the prohibitive risk of prostatectomy, such patients chose to use a catheter periodically, suffered with nocturia continuously and often were embarrassed by extreme frequency during the day. Now they submit to transurethral resection willingly. If any recent series of cases in which transurethral resection was performed is compared, with respect to age, with a series at the clinic in which prostatectomy has been employed, it will be found that ten times as many men older than 75 years of age are now seeking relief from urinary obstruction at The Mayo Clinic as there were during the days when the suprapubic method of operation was employed. It is obvious that the older a patient is the more advanced will be cardiac, renal and other organic impairment. Another factor in risk is the higher incidence of arteriosclerosis because the average age is greater. Table III shows the degree of arteriosclerosis, graded 1, 2, 3 and 4, encountered in the carefully analyzed series of 300 cases.

Renal insufficiency is the inevitable result of prolonged urinary obstruction and infection. The longer the period of obstruction and infection, the greater will be the degree of renal insufficiency. Many tests of renal function are employed but, from a practical standpoint, the estimation of the blood urea seems to be the most satis-

factory method of determining the degree of renal impairment. In most instances in which there was evidence of severe renal insufficiency according to the concentration of blood urea, other tests, such as estimations of the fractional elimination of phenolsulphonphthalein, determinations of the concentration of creatinine and serum sulfate and urea and sulfate clearance tests were performed. The degree of renal insufficiency encountered among the 1,200 patients is indicated by the following: in only 56 per cent of cases was the concentration of urea 40 mg. or less per 100 c.c. of blood; in 39 per cent the concentration of urea was 41 to 100 mg. per cent, in 2 per cent, 101 to 200 mg. per cent and in 1 per cent, 200 mg. per cent or more. In 2 per cent of cases the concentration of blood urea was not estimated; instead, a phenolsulphonphthalein excretion test was employed to estimate renal function.

The patient whose urinary obstruction is caused by carcinoma of the prostate gland has been particularly benefited by the development of the method of transurethral resection. Formerly, such cases were accorded permanent cystostomy, for it was the only method of relief available after the diagnosis had been made clinically. At the present time, transurethral resection in such cases can be performed at a risk no greater than that accompanying operation in cases of benign hyperplasia and relief of obstruction can be expected to endure for a few months or for many months. In instances in which subsequent resection is necessary, the second operation is more easily borne and is more acceptable to the patient than the inconvenience caused by permanent cystostomy. Patients for whom a second operation has been necessary have never accepted the alternative of permanent suprapubic cystostomy in preference to transurethral resection. Of the 1,200 patients studied, 16.4 per cent proved to have carcinoma of the prostate gland.

It is remarkable that, in spite of advanced age and associated cardiovascular renal disease and other complications, transurethral resection can be performed so safely that it was necessary to withhold operation in but very few instances. In those cases in which the patient was denied transurethral resection, his condition was such that the risk of any operation would have been prohibitive or, if transurethral resection had been successful, the life expectancy of the patient, because of the presence of other disease, was so small that the operation was not deemed justifiable.

PREOPERATIVE MANAGEMENT

The preoperative and postoperative management of these patients of advanced age in our opinion should be different from that given younger patients. If not, there is no question that the results of treatment will be inferior. The patient who is more than 70 years of age must be gotten out of bed each day; otherwise, several complications might develop. The lowering of blood pressure which occurs through continued rest in bed predisposes to arterial thrombosis and may result in cerebral or coronary accidents. It is a well recognized observation that patients confined to bed more than a day or two rapidly lose strength; this is especially true of the aged.

In the absence of renal insufficiency, we believe it is best to avoid prolonged urethral drainage by catheter. In order to comfort the patient and avoid repeated catherization, an inlying urethral catheter was used in the majority of cases until the general examination and laboratory studies were completed. Beyond a period of forty-eight hours preoperatively drainage by catheter was used only to reduce renal insufficiency or until associated conditions, such as cardiac decompensation or diabetes, were controlled. During this time, the patient is urged to stay out of bed most of the day unless cardiac decompensation or other conditions contraindicate it. The presence of an inlying catheter must not prevent the patient from being up and about.

In the presence of renal insufficiency, continuous drainage by urethral catheter is employed until the concentration of urea has become stabilized at a level which assures that operation can be safely under-

taken. If the patient is able to take generous quantities of fluid by mouth without the occurrence of nausea or vomiting, he is urged to drink 2,500 to 3,000 c.c. of fluid daily. When he is unable to take this amount, fluids are given intravenously as a supplement. Under ordinary circumstances 1,000 to 1,500 c.c. of physiologic saline solution is well tolerated if given slowly. However, care must be exercised in the use of solutions of sodium chloride in the presence of advanced renal insufficiency, owing to the fact that excessive quantities of salt may hinder recovery of renal function or may actually be harmful to the kidneys. Edema of the dependent portions of the body will develop if excessive quantities of salt are given. Under these circumstances, the use of a 5 or 10 per cent solution of d-glucose in distilled water is advisable. Attention has been called previously[1] to the value of employing alkali in the relief of acidosis incidental to renal insufficiency.

It must be remembered that patients of the older age group do not tolerate digitalis so well as patients of the younger age groups; therefore, this drug must be used with extreme care. Patients who are suffering from auricular fibrillation but whose cardiac rate is less than 100 beats per minute without evidence of cardiac decompensation have not been digitalized preoperatively. It has been found that, if digitalis is necessary postoperatively and is then given intramuscularly, the desired effect will be produced with a rapidity sufficient to meet the emergency.

Edema in cases of renal insufficiency secondary to urinary obstruction is rarely seen except as a result of cardiac decompensation or as a result of administration of excessive quantities of sodium chloride. The edema resulting from cardiac decompensation, in most instances, responds satisfactorily to the administration of salyrgan.

OPERATION

With very few exceptions we have used spinal anesthesia, injecting 50 to 80 mg. of procaine into the spinal canal through the interspace between the third and fourth lumbar vertebrae. It is unnecessary to

TABLE IV
AMOUNT OF PROSTATIC TISSUE REMOVED

Tissue Removed, Gm.	Per Cent of 1,200 Patients
Less than 5	10.2
5–10	17.3
10–20	30.2
20–30	17.0
30–40	8.9
40–50	6.9
50–120	9.5

inject a larger amount than this. The risk of operation among elderly patients varies in direct proportion to the time consumed, and it is therefore highly important that the operation should be of short duration. We prefer to complete it in two stages rather than prolong the transurethral resection at the risk of causing surgical shock. It is extremely important that a fall in blood pressure be prevented whenever possible, but if it should occur in spite of precaution, the pressure must be brought back immediately by injecting a solution of adrenalin or ephedrine. Loss of blood should be reduced to a minimum. Fall in blood pressure and excessive hemorrhage frequently result in renal insufficiency, oliguria and, sometimes, anuria. We have seen several cases in which restoration of blood pressure to normal and repeated transfusions have failed to benefit the patient after there has been a prolonged period of shock. The use of concentrated solutions of d-glucose and the derivatives of xanthine may be beneficial in such cases. If the operation is to result in eliminating the obstruction to the flow of urine, a large amount of tissue must be removed. It is not enough merely to cut a gutter through an enlarged middle lobe. A fair proportion of the entire gland must be excised. In these 1,200 cases, an average of 22.1 Gm. of tissue was resected; in 9.5 per cent of the cases 50 Gm. or more of tissue was removed in order to relieve the obstruction adequately. (Table IV.)

In the majority of the cases in this series the Thompson resectoscope was employed. The construction of this instrument is such

that many movements of the surgeon's hands which are necessary to the manipulation of resectoscopes of other types have been eliminated; hence, it is possible to resect a large amount of tissue in a short time. The removal of 2 Gm. per minute is not unusual. At the conclusion of the operation a hemostatic bag should be inserted in all cases unless a very small amount of tissue has been resected and it has been possible to control bleeding exceptionally well. The principal reason for this is that, in our experience, extreme fluctuations of blood pressure occur much more often among aged patients than among younger patients and every precaution possible against postoperative hemorrhage should be exerted.

TABLE V

POSTOPERATIVE MAXIMAL TEMPERATURE (1,200 PATIENTS)

Degrees F.	Per Cent
99 −	44.2
100	26.2
101	14.4
102	8.4
103	3.9
104 +	2.9

It should be unnecessary to emphasize that the operation be performed with strict aseptic precaution. The old teaching that the bladder and urethra in the large majority of cases are already infected and that there is therefore no need for aseptic technique is wrong. In our experience, postoperative febrile reactions will occur in inverse proportion to the aseptic care employed at operation. Extremely old patients are often greatly prostrated by fever; they are particularly likely to become delirious and irrational and as a result convalescence is prolonged. In 70.4 per cent of this series of 1,200 cases the temperature was never higher than 100°F. (37.7°C.) postoperatively; furthermore, in only 2.9 per cent did it go as high as 104°F. (40°C.). (Table v.)

POSTOPERATIVE MANAGEMENT

Getting the patient out of bed as soon as possible after operation is highly important. We believe that this point cannot be too greatly stressed. Our patients almost always are up on the day following operation. The catheter is removed in forty-eight or seventy-two hours, following which the patient must be observed very carefully. The catheter should be reinserted if there is the slightest indication that urinary obstruction still exists. Unless voiding is accomplished easily, fever, chills and severe complications are likely to occur. In cases in which there has been prolonged uremia, secondary anemia often will be found and it is generally wise to improve this condition by giving transfusions of blood, particularly if the amount of blood lost at operation and immediately afterward is more than the average.

In this series of 1,200 cases, the postoperative stay in the hospital was less than one week in 26.7 per cent of cases, and less than two weeks in 75.8 per cent, whereas only 9.5 per cent stayed more than three weeks. Thus, it can be seen that there are nine chances to one that the elderly patient will be out of the hospital within three weeks after operation, an average stay of much shorter duration than was ever experienced following the performance of suprapubic prostatectomy.

MORTALITY RATE

It is remarkable that as a result of careful management of these cases operation

TABLE VI

ANALYSIS OF ALL CAUSES OF DEATH: 1,200 PATIENTS, 1,361 RESECTIONS

Case	Age	Cause	Days Following Operation
1	85	Bronchopneumonia; aortic sclerosis	21
2	81	Pyelonephritis	32
3	76	Calcareous aortic stenosis; pyelonephritis	26
4	77	Aortic endocarditis; septicemia	24
5	71	Septicemia	9
6	77	Renal insufficiency; pyelonephritis	24
7	77	Pulmonary embolism	3
8	78	Septicemia (hemolytic streptococcus)	3
9	70	Coronary occlusion	1
10	83	Carcinoma bladder; gangrenous cystitis	12
11	74	Bilateral bronchopneumonia	17
12	76	Septicemia	12
13	81	Pulmonary embolus	2
14	78	Coronary occlusion	7
15	77	Lobar pneumonia	5
16	70	Mesenteric thrombosis	10
17	88	Multiple cerebral thrombosis	12
18	77	Multiple cerebral thrombosis	6
19	74	Shock; cardiac failure	1
20	73	Shock; coronary sclerosis	1

can be performed in this age group with a mortality rate of 1.6 per cent. Of the 1,200 patients, twenty died. A résumé of the causes of death is given in Table VI.

RESULTS OF OPERATION

If transurethral prostatic resection has been done properly, the functional results, both immediate and late, will be equal, if not superior, to those which can be obtained by any other type of prostatic operation. As a matter of fact, it is not unusual to admit a patient suffering from complete retention of urine, investigate his general condition by ordinary methods and, if it is found satisfactory, perform the transurethral operation within forty-eight hours of his admission. Two or three days later drainage by urethral catheter is dispensed with and the patient usually is able to void a large stream painlessly and, furthermore, he is able to empty the bladder completely; this may be proved by catheterizing the bladder to determine the absence of residual urine. Within a week from the day of admission more than a fourth of the 1,200 patients of this series had been dismissed from the hospital. Thereafter they reported at the office, where they were observed daily until a fortnight or so after operation, at which time they were dismissed from our care. Such a result is ideal and cannot be obtained when there are complicating factors such as severe uremia, cardiac decompensation, diabetes or advanced anemia all of which require preoperative treatment. Such patients do not recuperate from operation as quickly as those who do not have such disease and must be watched carefully in order to prevent the occurrence of complications postoperatively.

The urine, which is always cloudy immediately after removal of the catheter, generally clears progressively until, at the time of dismissal, it is only slightly hazy and, sometimes, is clear on gross inspection. In some cases, severe deformity of the bladder such as extreme sacculation, diver-ticulation or deep cellule formation and renal deformity, such as hydronephrosis and hydroureter, complicate the case and delay the elimination of pus from the urine. Such patients, if they empty the bladder properly, will not suffer untoward symptoms and, after a few weeks, the voided urine will be much clearer than it was prior to operation. Certainly, in our experience, the urine even in such cases became clear much more quickly than it ever did in similar cases following suprapubic prostatectomy. We do not administer urinary antiseptics routinely, with the single exception of methylene blue. This drug seems to have slight hemostatic properties and is an excellent postoperative medication. Any slight degree of urgency or any tendency toward incontinence will, because of soiling of the clothing, be called to the patient's attention and, hence, will be reported. Fortunately, this occurrence is extremely rare and, as a general rule, quickly subsides. In our experience, incontinence occurs less often after transurethral resection than it does after suprapubic or perineal prostatectomy.

It can be expected that nocturia will be relieved promptly; in many cases in this series, patients have reported sleeping through the night without voiding, within two weeks after operation. In a small percentage of cases, moderate nocturia will persist, often in part because of a large nocturnal secretion of urine by the kidneys. Even in such cases, if transurethral resection is to be regarded as successful, it must cause a definite decrease in nocturia. In our opinion, a complete analysis will force the conclusion that, without any selection of cases, the results of transurethral resection at the hands of an experienced surgeon are superior to those obtained by either suprapubic or perineal prostatectomy.

REFERENCE

1. HABEIN, H. C. The relation of water, sodium chloride, and acid-base balance to renal function in the treatment of lesions of the urinary tract. *Am. J. Surg.*, 38: 6–17 (Oct.) 1937.

RESECTION VERSUS PROSTATECTOMY

THOMAS J. KIRWIN

NEW YORK CITY

MUCH has been written about surgery of the prostate. The literature of the resection method is especially voluminous, and is often highly controversial in tone. No busy surgeon could possibly review it all. What he does read is likely to leave him with the confused impression that enthusiasm *for*, or prejudice *against* the resection technique, has made the authors lose sight of the main objective of prostatic surgery, namely, *relief of urinary obstruction.*

Free drainage from the bladder is the sole purpose of all operative intervention upon the prostate—whether by suprapubic or perineal prostatectomy, resection or nonoperative methods. The question is not: "What is the best method to adopt for *all* cases of prostatic hypertrophy?" but rather: "How can the urinary obstruction of *this* patient best be relieved?"

Until recently the prostatic surgeon had choice of but two ways to accomplish this purpose: (1) perineal prostatectomy, a very ancient operation, having its beginnings in the lithotomy procedures of the Middle Ages; and (2) suprapubic prostatectomy, a relatively "new" procedure, first done in this country by Fuller in 1895.

Perineal Prostatectomy. The past thirty years have seen great improvement in both these methods. Young's perineal operation, performed with his specially designed instruments, was originally worked out with such technical precision that its details have remained unchanged for more than a quarter century. It requires a trained "surgical team" and will always remain an "expert's operation." But those capable of performing it will have good results—low mortality and permanent functional improvement.

Suprapubic Prostatectomy. Because suprapubic enucleation seems a "simpler" procedure, a great many general surgeons, without special urologic training, have not hesitated to attempt it. This is probably the chief reason why the suprapubic procedure is generally credited with a higher mortality rate. As cystotomy usually precedes it, the idea of making a single incision serve for both stages of the intervention appeals to both surgeon and patient.

Prostatic Resection. The introduction of a third method—intraurethral resection—inevitably altered the surgical viewpoint. All intraurethral procedures are largely dependent upon mechanical aid. The skill of the operator is restricted by the tools he is forced to use. At present several different types of resectors are available, with any one of which it is possible to attain excellent technical results *after sufficient practice.*

The indications for intraurethral resection are wider than for the older methods. Many patients for whom open operation is out of the question can be relieved when intervention is by way of the urethra. The shortened hospital stay is economically attractive to most patients, as well as to the hospitals themselves, because the rapid turnover of ward cases lessens the cost of caring for each patient. Thus—though they should not—financial considerations inevitably influence the choice of operative procedure.

Believing resection to be a "simpler" operation, younger men are now seeking treatment. For the same reason an ever-increasing number of "old" cases clamor for relief—conditions of long standing in men of advanced years. Debilitated patients, and those weakened by organic derangements, renal, cardiac and vascular, who are unable to endure open operation or seem to think they cannot because of its attendant hazards, may often be relieved

by this means. This, together with newspaper propaganda, and the laity's belief that it is a "minor" procedure, steadily increases the number of resections.

Malignancy. Unquestionably, resection has proved an excellent means of handling many cases of prostatic cancer, but as a diagnostic aid for the detection of malignancy it leaves much to be desired. Tissue removed by the cutting electrode does not lead itself to the preparation of sections so well as that secured by the scalpel. Not only is it difficult to differentiate cell structure which has been in contact with the electrocautery, but also the fact that questionable tissue is frequently left behind, throws suspicion upon a negative pathologic report, inasmuch as malignancy may be present in tissues which were not resected. Even though the estimate that from 15 to 30 per cent of hypertrophied prostates are actually malignant is too high, as Moore of Cornell contends, nevertheless all these considerations are of great importance.

Nonoperative Methods. Because surgeons are now operating for wider indications than ever before, they are undoubtedly overlooking the possibilities of nonoperative treatment, which they used to consider before performing suprapubic or perineal prostatectomy. Dilatation, tissue shrinkage, or—in frankly malignant cases—radon implantation, could undoubtedly take the place of many resections. Yet how is the conscientious surgeon to judge?

Degrees of Hypertrophy. First of all he should consider the size of the gland in question. Teem (1936) gives the average measurements of the normal adult prostate as: length, 3.08 cm.; width, 3.84 cm.; depth, 2.7 cm.; and weight, 18 to 23 Gm. Therefore, in a prostate removed for hypertrophy which is found to weigh from 250 to 300 Gm., the enlargement is generally designated as "moderate," but when it reaches 400 to 600 Gm. it may justifiably be termed "enormous." It is generally conceded by surgeons that these "enormous" prostates should be treated by open operation unless such surgery is absolutely contraindicated. These huge hypertrophies now seem to be more uncommon, possibly because patients are coming under treatment earlier. Therefore, at many clinics resection has become the rule, prostatectomy the rare exception.

Advantages of Open Operation. Yet the perineal exposure gives far better vision than is possible even with the best resection instrument, and areas suspected of being malignant readily provide tissue for biopsy. If cancer be found the operation can be changed to a radical removal. Moreover, perineal prostatectomy can be done in a fraction of the time required for intraurethral resection of even moderate hypertrophies, and never imposes the shock and strain undergone by a patient who must remain much longer on the table while the prostate is being removed in small pieces than he does when the mass is excised entire.

Knowledge of All Methods Desirable. My position is that every case must be considered individually. The day is past when the urologist could perfect himself in the technique of one particular method of prostatic removal, disregarding the claims of all others. It is an absurd assumption that certain surgeons can perform only a particular type of operation on the prostate. Every director of a urologic service owes it to his students to instruct them in the technique of all three methods and to teach them to evaluate each, suprapubic, perineal or intraurethral resection, according to the conditions presented by the individual case. Without such training no operator will ever be able to satisfy either himself or the majority of his patients. Resections will continue to be done in ever-increasing numbers—there is no doubt of that—but every now and then we shall encounter patients whose condition will not permit the use of any resecting instrument. We must also consider the 15 to 30 per cent of malignant cases. For some of these patients resection is efficient after diagnosis has been established, yet many will come

in the "borderline" where the surgeon's individual judgment must be final, for no hard and fast rule can be laid down. Even if younger men are now applying for treatment there is no reason why the number of resections should be so greatly increased. Nonoperative measures, such as dilatation, prostatic massage, or tissue "shrinkage," are even more effective in these "early" cases than in those where a marked degree of hypertrophy already exists.

CONCLUSION

I wish to emphasize that nonoperative methods, such as gradual dilatation by sounds or the Kohlman dilator, or the "shrinkage" method which I have advocated, should still be kept in mind, and never be entirely discarded for intraurethral surgery. We should not resort to resection at the first indication of middle or lateral lobe intrusion.

It is well to keep in mind the distinction between different types of prostatic hypertrophy set forth by Alexander Randall: "If there be large intravesical intrusion of hypertrophied lobes, the suprapubic approach should be employed and the tissues within the bladder enucleated. If there be marked extravesical intrusion, with large masses of tissue in the posterior urethra, enucleation by way of the perineum is in order. But for relatively small lobes, causing urinary obstruction by position rather than size, intraurethral resection is by far the most efficient method.

REFERENCES

Alcock, N. G. Prostatic hypertrophy. *J. A. M. A.,* 104: 734, 1936.

Baker, W. W. Perineal prostatectomy. *Ann. Surg.,* 104: 424, 1936.

Bumpus, H. C., Jr. Transurethral resection; its indications, limitations and complications. *Surg., Gynec. & Obst.,* 64: 324 (Feb. 15) 1937.

Davis, T. M. Prostatic resection. *Urol. & Cut. Rev.,* 39: 372, 1935.

Kirwin, T. J. Types of vesical neck obstruction suited to intraurethral resection: advantages of treatment by the rotary prostatic resectoscope. *Am. J. Surg.,* 19: 1 (Jan.) 1933.

Kirwin, T. J. Treatment of prostatic hypertrophy by a new "shrinkage" method. *J. Urol.,* 32: 481, 1934.

Laidley, J. W. S. A survey of prostatic surgery today. *Australia & New Zealand J. Surg.,* 6: 324 (July) 1936.

Lowsley, O. S., and Kirwin, T. J. Suprapubic prostatectomy. *J. A. M. A.,* 97: 1669 (Dec. 3), 1931.

Mayo, C. H. Treatment of the enlarged prostate gland by modern methods. *Texas State J. Med.,* 32: 221 (July) 1936.

Orr, Louis, and Rue, D. T. Present day conceptions of the management of prostatic obstruction. *J. Florida M. A.,* vol. 22, July 1936.

Randall, Alexander. Discussion of paper by Bumpus.

Teem, M. V. Size and weight of normal and of pathologic prostate gland. *Arch. Path.,* 22: 817 (Dec.) 1936.

Young, H. H. Transurethral resection. *J. A. M. A.,* 102: 1913 (June 9) 1934.

Young, H. H. Transurethral Resection of the Prostate. Nelson's Loose Leaf Surg., Vol. v, p. 300, 1937.

STANDARDIZATION OF A BLOOD TRANSFUSION SERVICE*

STANLEY E. MONROE, M.D.

Clinical Assistant in Surgery, Northwestern Univeristy Medical School

EVANSTON, ILLINOIS

THE transfusion of blood is now such a common procedure that every hospital does well to simplify and standardize its methods. Although methods and their desirability vary from hospital to hospital depending upon equipment, past experience, and personnel, the citrate method is, perhaps, the most widely used.

The preference for this method at the Evanston Hospital is shown in Table I, based on blood transfusions given from July 1, 1936 to June 30, 1937. These transfusions were given to ninety-six patients, fifty-two of whom were female, forty-four male. Distribution into groups is given in Table II.

Interns and residents gave 114 transfusions, attending men 68, while 14 were given jointly by intern and resident staff and attending men.

The reactions are shown in Table III. No reaction was a threat to a patient's life. No error in a compatibility test occurred. There was no lengthening of convalescence due to a reaction.

TRANSFUSION TEAM

The transfusion team is made up of the surgical resident and one of the senior surgical interns. The resident in surgery is responsible for the proper instruction of these men. In his absence the two surgical interns on the senior service constitute the transfusion team.

THE CITRATE METHOD

Inasmuch as the citrate method meets with greatest favor in our hospital we have endeavored to make it as easy and safe a procedure as possible. The equipment we describe is not original with us. Similar equipment is used in a number of hospitals, and Wenzel and Howard[1] published in 1936 a method of blood transfusion not unlike that described here. We favor a closed container for the blood to prevent contamination. Provision for suction is sometimes of value. It is always used by us to introduce citrate solution into the system.

The container for the blood is a flask of the kind used for intravenous fluids. We have taken out the rubber stopper and have fitted it with a piece of glass tubing reaching just below the 500 c.c. mark. For a strainer two or three layers of coarse mesh

TABLE I

	Number	Per Cent
Transfusions given	198	
Citrate method	178	90
Percy tube	10	5
Multiple syringe	8	4
Brook's tube	2	1

TABLE II

Group (Moss)	Number	Per Cent
I	0	0
II	43	44 8
III	11	11.5
IV	42	43 7

TABLE III

	Number	Per Cent
Reactions—all kinds	14	7.0
Urticaria without chills and/or fever...	3	1.5
Chills and fever.......	11	5.5

gauze are placed over the under side of the rubber stopper and caught around its edge as it is fitted into the flask. The flasks are

attention to all superficial veins in the region of both cubital fossae. Failure to select the vein easiest of access often makes a difficult
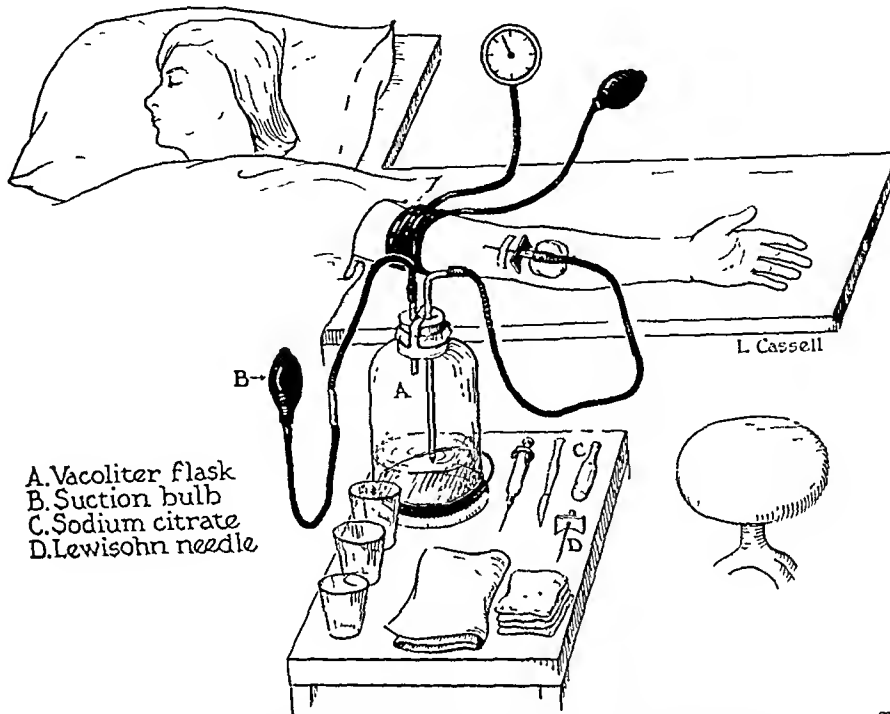
FIG. 1. Method of withdrawing blood from donor. The sphygmomanometer cuff is inflated to approximately the level of diastolic pressure.

A. Vacoliter flask
B. Suction bulb
C. Sodium citrate
D. Lewisohn needle

graduated by applying paraffin, scratching it off at 100 c.c. levels and etching with hydrofluoric acid. A rubber tube 12 to 14 inches long and fitted with an adapter for a cannula is fitted to the glass tube in the stopper. A similar rubber tube of greater length, about 20 inches, is fitted with glass adapters, one of which is to be placed in the rubber stopper and the other to serve as attachment for the two-way bulb.

When the apparatus is thus assembled, the citrate solution is placed in a glass containing 1 ounce of normal saline, the solution is then drawn into the system through the cannula that is affixed to the intake tubing. The negative pressure is furnished by the suction bulb.

Skilful technique in venipuncture demands considerable training. In our experience, observation of the following points will aid in better results:

Before attempting to draw blood from the donor, examine both arms and pay

task of what should be an easy one. A proper tourniquet is essential; the compression band of a sphygomomanometer is unexcelled, for by use of it one can make certain of the degree of constriction at all times. It is easily adjusted, comfortable, and may be applied with the cuff folded lengthwise so that it takes up but little room.

Novocaine is always used. A wheal is suitably placed alongside the chosen vein. A small cut in the skin, parallel to the lines of cleavage is made to admit the needle. Sharp needles with proper bevels are important. The rate of flow from the donor's vein is little influenced by the direction of the needle. As it is usually more convenient to insert the needle centrad we usually do it that way. The needles should be clean to prevent any tendency to clot in the needle. At the Evanston Hospital Lewisohn and Kaliski needles have proved most practical. (Fig. 1.)

For use in the recipients 19-gauge needles most often are chosen. Normal saline is used to start the venoclysis. This is
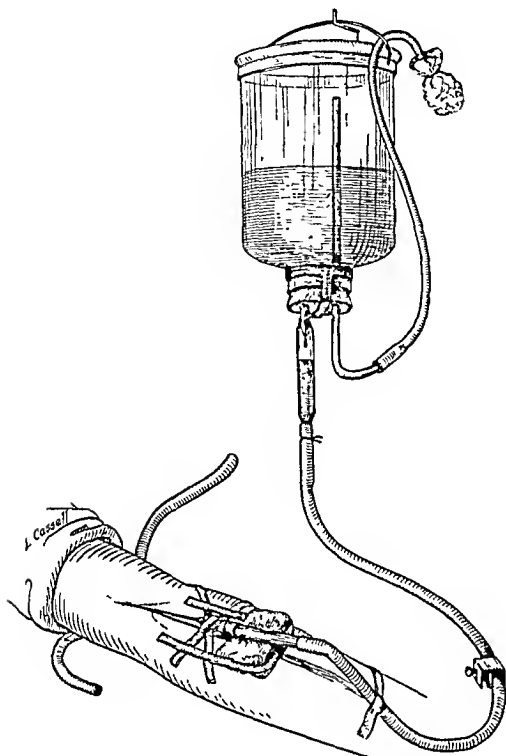
FIG. 2. Method of administering citrated blood.

done before the blood is taken from the donor. One should only rarely need to expose a recipient's vein, simple venipuncture is easier and safer. Again, one should examine the superficial veins so to choose that the venipuncture be as easy as possible. Nearly all patients have suitable veins on the dorsum of hands and wrists and forearms. These veins are not difficult to use and allow the patient considerable freedom of his arm. Two points that we

have found helpful in using the veins on the dorsum of the hand are the following: The hand should not be bound down to an arm board or pillow before the venipuncture is done. If the hand and wrist are left free, one can, by flexing the hand, put the vein on just the optimum amount of stretch. Once the needle is inserted the hand and wrist should be placed on a support. When the veins do not readily fill, we place the hand and forearm in a basin of water as warm as can comfortably be borne, gently massaging the fingers and hand the while. When, with the tourniquet properly set, the vein fills well one's task is made easier. Soft, flexible rubber tubing of small bore, such as is commonly used for blood pipettes, is more efficient as a tourniquet than the large tubing often used, for with the latter, tucking under the ends may make a tunnel for the vein or its collaterals. The tourniquet should always be applied where it will do the most good and this is usually only a few inches above the section of vein one wishes to use.

One-half per cent novocaine is used to make a wheal in the skin when starting the intravenous in children and nervous adults. The introduction of the needle is facilitated if the relationship of the needle bevel to
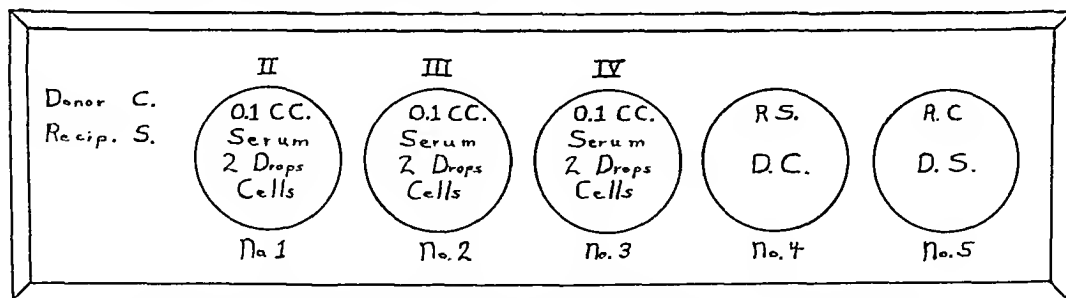
FIG. 3. Diagram of Coor's porcelain spot plate showing use in blood grouping

ture is easier and safer. Again, one should examine the superficial veins so to choose that the venipuncture be as easy as possible. Nearly all patients have suitable veins on the dorsum of hands and wrists and forearms. These veins are not difficult to use and allow the patient considerable freedom of his arm. Two points that we

the vein is kept in mind. One can keep the bevel side located by placing it on the calibrated side of the syringe. In pushing the needle through the skin no immediate attempt is made to enter the vein. The needle is brought alongside the vein, then in a separate motion is slowly pushed into it and worked along inside the lumen a

distance of about ½ inch. In inserting the needle the bevel is held against the side of the vein. When the vein is poorly filled or is

once it is entered may offer difficulty. At this point one can get considerable help by aspirating 1 c.c. or a little more blood into

---

Form C-23 1M 8-36

EVANSTON HOSPITAL ASSOCIATION

Record of Blood Transfusion

Place in patient's chart immediately preceding progress notes.

Hospital No._____

Patient's Name ....

Compatibility Tests:
     Patient is group_____Moss. Grouped by_____
     Donor is group_____Moss. Grouped by_____
     Cross Match: D.C. - P.S._____
                 P.C. - D.S._____   Matched by _____

Donor: Relationship_____ Friend_____ Professional_____

     Name .... Age____   M S W D   Number Times
                                                                    donor before _____
     Address .... Tel..............

     Hb._____Date_____ Wasserman_____Date_____ Kahn_____ Date_____

                      Date Registered at E. H._____

TRANSFUSIONS: Date_____ 19_____ Time_____ A.M. P.M.

     Given by Dr.. Asst..

     Reason for Giving _____.

     Previous Transfusions_____

     Method used _____

     Time Consumed in Actual Transfer of Blood_____

     Amount of Sod. Citrate used in Grains_____ Other Solutions_____

                   Amount of Blood Given_____

     Method of Entry Into Veins:
         (a) Donor-Surgical Exposure-Yes   Direction of Needle: Centrad
                         No   Distad
         (b) Patient-Surgical Exposure-Yes
                         No

     Condition During First 10 min. of Transfusion_____

     Reaction During or After Transfusion

         Probable Explanation:

	Nurses' Record
Time	_____
T.	_____
P.	_____
R.	_____
BP	_____

Use Reverse Side of Sheet For Additional Data.

Fig. 4. Form for record of blood transfusion.

---

small, scarcely larger than the needle, introducing the needle further into the vein

the syringe, and then while the tourniquet is still set, reinjecting the blood, the needle

being gently pushed along inside the lumen which is at the time distended.

When called upon to start fluids intra-

FIG. 5. Blood donor certificate.

venously on a patient in shock, it is often difficult to get a vein properly filled. Occasionally the tight spiral bandaging of a hand and ascending up the forearm, with a tourniquet tightly constricting the arm, will fill a superficial vein at or just below the cubital fossa and make the insertion of the needle relatively easy. When the intravenous administration of saline solution has been started in the recipient, the blood is drawn from the donor into the flask as described above. Then this flask is wrapped in a sterile towel and carried to the recipient's room where it is substituted for the flask of saline by changing the drip chamber from one to the other. One should be certain that the administration of citrated blood is not preceded or immediately followed by Ringer's solution or other calcium-containing solutions. Calcium ions made available in this manner will cause immediate clotting. In our experience we have had no untoward results with the use of blood and other intravenous fluids when given over a considerable range of room temperatures. We prefer that they be given at body temperature. (Fig. 2.)

The technique for blood typing and compatibility used at the Evanston Hospital is the macroscopic porcelain plate method (modification of Brice). It was introduced here by our pathologist, Dr. Eustace Benjamin, and I am indebted to him for the description which follows.

Five c.c. of blood are withdrawn in the usual manner from the median basilic veins of the recipient (patient) and the prospective donor. Three drops of blood from a 20-gauge needle are placed in 4 c.c. of a physiologic saline solution containing 2 per cent sodium citrate. This serves as a cell suspension. The remainder of the blood is put into a 15 c.c. centrifuge tube and allowed to clot. After centrifugation the supernatant clear serum is used for compatibility and serologic tests.

A Coors porcelain spot plate is used for performing the test. Into each of the first three hemispherical concavities 0.1 c.c. of pooled type serum is placed. Type II (Moss) is placed in concavity No. 1, Type III in concavity No. 2, and Type IV in concavity No. 3. To each of these are added two or three drops of the red cell suspension to be typed. Fifteen minutes is the minimum time permitted to elapse before the reading is recorded (See Fig. 3), although agglutination or clumping usually occurs in a few minutes. The plate is then lifted with the hands and the cell suspension-serum mixture is thoroughly mixed by oscillation and rocking of the plate for several minutes.

Red cell clumping occurs in all type sera wherein the type serum does not correspond to the type of the red cells in the mixture. Type determination is indicated by absence of agglutination. There is an even settling of the cells in the bottom of the hemispherical concavities.

Compatibility to Direct Matching. Two parts of recipient's serum and one part of donor's cell suspension are placed in concavity No. 4 and labelled with a wax pencil R.S.-D.C. Two parts of donor's serum and one part of recipient's cell suspension are placed in concavity No. 5 and labelled R.C.-D.S. These mixtures are appropriately mixed. If at the end of thirty minutes no clumping of the cells has occurred in either preparation, the bloods are compatible. If at any time clumping occurs in R.S.-D.C. or in both concavities, the bloods are incompatible.

Advantages of This Method. Rapidity of performance: six tests may be performed at the same time, including typing and direct matching. Absence or presence of clumping is noted with ease and certainty. We have performed 1,722 tests with this method during the past year without error.

MOSS CLASSIFICATION OF BLOOD GROUPS

Cells	Serum		
	II	III	IV
I	x	x	x
II	o	x	x
III	x	o	x
IV	o	o	o

x agglutination
o no agglutination

Good record keeping is important to a blood transfusion service. Forms for the purpose need not be elaborate, but should be carefully planned. We have designed a form (Fig. 4) to give a summary of the transfusion, patterned after one by Martin and Whyte.[2] This sheet is carefully filled out as each transfusion is finished. A small form (Fig. 5) is used for recording data on donors who are typed for the patient. These blanks are filled out in triplicate by the technician who does the tests. One copy is given the prospective donor, one is placed in the laboratory files, and one is available to the surgeon who is to give the transfusion. This system has proved itself free of several types of bookkeeping errors which may cause mistakes when intermediaries convey information from the technician to the surgeon.

The Evanston Hospital maintains a private donor list made up of medical students, townspeople, and a few hospital employees. The giving of blood by interns and employees of the hospital is discouraged. Every member of the donor list is given a Kahn test and is carefully questioned about diseases which would make him ineligible as a donor.

SUMMARY

1. A record is presented of 198 blood transfusions done at the Evanston Hospital in the year ending June 30, 1937.

2. Suggestions are made for the administration of citrated blood. A method of typing and matching bloods is detailed.

3. Record forms which have proved useful are reproduced here.

REFERENCES

1. Wenzel, J. F., and Hammer, H. J. New method of blood transfusion. *Am. J. Surg.*, 34: 119 (Oct.) 1936.
2. Whyte, Martin E. Organization of transfusion service and blood donors' bureau for average hospital. *Am. J. Surg.*, 32: 439, 1936.

FIRST AID AND TRANSPORTATION OF SUSPECTED SPINE INJURIES

JAMES E. M. THOMSON, M.D., F.A.C.S.

Chief, Bone and Joint Surgery, Bryan Memorial Hospital; Attending Orthopedic Surgeon, Lincoln General and St. Elizabeth Hospitals

LINCOLN, NEBRASKA

THE fact that most essayists on the subject of fractures of the spine avoid the very important phase of first aid and transportation in the treatment of injuries and suspected injuries of the spine has prompted this presentation. I firmly believe that the care or carelessness with which such victims are first handled bears a definite relationship to the ultimate amount of their disability and competence in adjusting themselves in their former or new occupations. Perhaps the reason why so little is said about the early handling of such injuries may be the fact that there has been such a variance in opinion as to which method is most appropriate.

Among the other factors that have prompted this report are: (1) the increasing number of spine injuries, due to automobile, farm and home accidents, as well as those contributed from industrial sources; (2) that since the victims of such injuries are usually first seen and moved by laymen, and since the first movements of these victims may injure or aggravate the already injured spinal cord, causing irreparable damage and paralysis and making functional recovery impossible, a unified effort is now in order to teach not only the physician, but more particularly the lay public, the grave consequences of misdirected, unintelligent handling of suspected spine injuries, and also to give an approved method by which such injured persons may safely be handled or moved.

The method herein suggested offers nothing new except that instead of recommending rather opposing procedures for spine fractures of the trunk and cervical region, it endeavors to adapt a single uniform procedure for all fractures of the lumbar, dorsal and cervical spines. Such an adaptation, or single procedure, seems essential, since it scarcely seems fair to ask the ordinary layman giving first aid under stress of excitement, or even the trained first aider or ambulance team, to be responsible for a diagnosis. They cannot always know whether the injury is in the lumbar or cervical region, except, of course, where evident paralysis exists. Where there is paralysis, the first aid attendant should recognize that if the victim cannot move his fingers, his neck has been broken; if he can move his arms and not his legs, the dorsal or lumbar spinal cord has been injured. But to go further than this in differentiation of the site of injury is presumptive. Symptoms are of little value, as the injured person may suffer severe pain and bruising in one portion of his back but have grave bony injury in another.

However, we may assume that if the character of the accident is such that a back injury might have occurred to the victim, or if the back, trunk, neck or head is the site of pain or evident violence, or if the patient is unconscious—he deserves well-directed first aid by an approved method of handling such injuries. Every man, woman and child should know how to proceed in an intelligent manner when such an occasion arises. Admittedly the finding of paralysis is uncommon, but it is to prevent damage to the cord by thoughtless bystanders that we urge that an approved single method of handling of all spine injuries be broadcast to laymen and practiced by physicians.

An extensive personal study, nationwide inquiry among physicians doing fracture work, and the suggestions of various frac-

ture committees have brought rather conclusive deductions with respect to an appropriate method of moving and trans-

tion and traction are essential. "Splint them where they lie."

Out of 117 thoughtful and well-directed

FIG. 1. Half-rolled blanket in front of victim. *A* supports the head with hand placed under the head from behind, so that slight traction can be maintained in the axis of the body. *B* places hands firmly on shoulder and hip. *C* takes charge of the legs.*

FIG. 2. Victim on his side with legs extended. With his free hand *A* extends the lower arm above the victim's head. The upper arm is also extended and the forearm flexed so as to support the head. The blanket is tucked against the body.

porting patients with spine injuries. The same basic principles apply here that apply to fractures of long bones. The crunching of bone fragments into each other and into the soft tissues causes injury and shock. Therefore, early and adequate immobiliza-

replies to the question of transportation of injuries to the lumbar and dorsal spine, forty-nine advised gently rolling, skidding, or with a blanket lift, placing the patient in the prone position on a stretcher, board, gate or door. Fifty suggested simply keep-

* The prone blanket lift is demonstrated by officers of the Nebraska Safety Patrol, herein designated as *A, B, C.*

ing the patient in a recumbent position on a board or stretcher. Of these, twenty-seven suggested that muscle splinting was sufficient to prevent further injury to the cord, while twenty-three advocated hyperextension by means of a pillow under the lumbar spine. Eighteen suggested the use of the Abbott frame as a part of an ambulance accessory equipment, and of these twelve suggested its use in hyperextension. There were twelve replies so fantastic, or individual, that they were not considered.

Fig. 3. The victim is rolled face down onto the blanket. The head is maintained in the same axis with the body.

Fig. 4. A and B grasp the blanket 6 inches lateral to the body, one hand on a level with the victim's elbows and the other with the hips. C grasps the blanket on either side of the ankles.

When it came to offering recommendations for the transportation of fractures of the cervical region much originality was displayed in the various methods advocated in the ninety-six replies. Fifty recommended gentle handling, placing on a hard

surface supine, with everything from sand-bags, bricks, hot water bottles, stuffed paper bags, rolls of clothing and shoes on

sion. The important factors pointed out, however, are: That the head or trunk should not be flexed or lifted forward towards the

FIG. 5. The lift and carry.

either side of the head, to a U-shaped sand-bag about the head, or a creased pillow. Twenty-nine recommended extension and traction, held by various means, throughout the period of transportation. Twenty-five recommended a collar of cotton; cardboard and cotton; cloth bandage; or clothing, such as a sweater, coat, shirt or a stuffed woman's stocking. There were four who advised a pillow under the shoulders to hold the head back in hyperextension. Four others simply suggested prevention of all movement, and there were four who thought that muscle splinting was sufficient. Only a few suggested methods of getting these patients onto the board or stretcher, and the importance of maintaining the cervical region in the same horizontal plane or axis as that of the spine of the trunk. I assume that their appreciation of the importance of this phase of the problem was implied with the recommendation for immobilization during transportation.

Every suggestion had its unique virtue. Not one is in itself applicable to every occa-sitting position, as these movements cause jack-knifing of the spine, crunching of the injured bony bodies and processes, increasing deformity and the possibility of spinal cord injury. Gentleness in handling, rolling or moving the entire spine from head to hips in the same horizontal plane or axis is essential, except as guarded hyperextension of the spine becomes a factor in the procedure. The minimum of movement of any kind is vital.

From these suggestions, and others, has been developed the prone, three man blanket lift, with head supported. This procedure seems adequate for the transportation of all suspected injuries of the spine, whether they are in the lumbar, dorsal or cervical region, and eliminates all diagnostic confusion or deductions on the part of the untrained. It requires little movement and is simple in accomplishment. It can be employed whether the patient is to be transported by ambulance, on a door, board, or on the floor of a truck, sedan or wagon, or whether a stretcher or cart, board, door or ladder is used as a

litter. It requires no special equipment. A blanket is usually available in a car, garage, filling station, nearby home or in

FIG 6 If conscious, the patient can steady his head by placing his hand on his occiput during transportation.

the ambulance. It maintains in extension not only the trunk, but also the cervical region. With the arm placed carefully under the forehead there is no possibility of jack-knifing in this region. It is a comfortable posture and enables the patient to breathe freely.

The New York-Brooklyn Regional Fracture Committee considered the prone position in relation to injuries of the extremities, abdomen, chest and head, and in doing so has consulted with fifteen general surgeons. The consensus of opinion is that the above proposed method is not harmful to the associated internal injuries or those of the extremities.

Technique. Three men, designated as *A*, *B* and *C*, approach the victim with a blanket. *A* supports the head with one hand placed under the head from behind so that slight traction can be sustained. *B* and *C* half roll the blanket in front of the patient and place the half-rolled blanket in close proximity to him. *B* then places a hand firmly on the shoulder and hip while *C* takes charge of the legs. The three men carefully roll the patient onto his side, turning the head, body and legs together. The half-rolled blanket is drawn close to the patient. *A* now extends the lower arm above the patient's head with his free hand.

The upper arm is also extended above the patient's head and the forearm flexed to support the head. The patient is then rolled forward onto the blanket which is then unrolled. The forehead now is supported on the flexed forearm of the patient, the hand resting just above the elbow, which is then flexed on the blanket. If the patient is conscious he can support his occiput with the free hand, or, if he is not conscious, fixation of the head and neck can be maintained by *A*, or small sandbags can be placed on either side of the head. *A* and *B* then place themselves on opposite sides of the patient and grasp the blanket about 6 inches lateral to his body, one hand being at the level of his elbows and the other hand at the level of the hip. *C*, meanwhile, grasps the blanket on either side of the legs just above the ankles. The three men lift the patient together, allowing the back to extend slightly. If a suitable conveyance is not at hand the blanket is wrapped about the patient until preparations are completed.

After careful first aid attention and transportation of the patient, it is difficult to estimate the amount of damage that can be done when on reaching the hospital unnecessary moving, improper handling and turning of the patient takes place. Once the patient has been placed in the prone position on a blanket, this position should not be changed until a diagnosis has been made and treatment instituted. His first stop should be the x-ray laboratory where postero-anterior and lateral x-rays are made while he is in the prone position. Clothing can be cut away or carefully lowered or raised as seems expedient. From here the patient is taken directly to the operating room where permanent treatment is begun.

There are those who believe that muscle splinting is sufficient to prevent further damage, and it is reasonable to assume that here such splinting is more effective than in the case of fractures of the extremities. Nevertheless, patients with back injuries should be adequately guarded against

further insult to the important structures involved. It would seem, therefore, rational to maintain the prone position until diagnosis and permanent treatment have been instituted.

SUMMARY

1. The care or carelessness with which those who have injuries of the spine or injuries of the back are handled, bears a definite relation to the ultimate amount of their disability.

2. Modern life and activity have increased the incidence of severe spine injuries to the extent that not only every doctor, but every layman as well, should recognize a common method of administering first aid and transportation of such victims.

3. A method is herein suggested which relieves the first-aider of the responsibility of making a diagnosis as to whether the injury is in the cervical, dorsal or lumbar spine.

4. It is a simple method requiring minimum apparatus and little practice.

5. It involves but a single principle—that is, rolling the victim as one would a log, "en masse," with the entire spinal column in the same horizontal plane, to a prone position onto a blanket.

6. Once in the prone position the patient should remain so until x-rays are taken and permanent treatment instituted.

"It is the fractured skull which is exhibited in our museums, not the damaged brain. . . . But, . . . what has happened to the skull is of far less moment than what has happened to the brain, and this has been given less study." (Cushing)

From—"A Textbook of Surgery," edited by Frederick Christopher (Saunders).

HIP INJURIES: SOLUTION OF THE "UNSOLVED FRACTURE"

James A. Jackson, M.D.   AND   J. Newton Sisk, M.D.

The Jackson Clinic

MADISON, WISCONSIN

WITH all due regard to our early preceptor, Royal Whitman, probably the first to put the treatment of fractures of the neck of the femur (the "unsolved fracture") on a scientific basis, his method or any other that requires prolonged confinement in bed in a body cast or any fixed form of external splinting, is passing into obsolescence for any but exceptional cases. Enough cases are now on record to show that internal fixation provides a much higher percentage of union, lower incidence of stiffening of the knee joint and other complications, and lower mortality rates. In addition, the patient is happier and his hospital stay is much shortened.

For many years we have been firm advocates of the internal method of fixation in certain fractures. Since Smith-Petersen, some seven years ago, first devised his three-flanged nail for fixation of hip fractures, there has been increasing interest on the part of all fracture men in this form of treatment. Although many types of screws, nails, and pins have been devised by different surgeons, we believe the Smith-Petersen nail, in its present perfected form, is superior both in its mechanical efficiency and in the simplicity with which it is applied.

Experimental work is now being carried on with different metals in an endeavor to find a combination which will have the requisite tensile strength and hardness, be non-corrosive, and give the least irritation to body structures.[1] It is probable that in the near future we shall have an alloy of vitallium in which vanadium is substituted for the chromium. This alloy will fulfil these requirements. When the metallurgists solve the problem, the Federal Bureau of Standards should require the instrument makers to conform to this formula in their manufacture of nails, plates, screws, etc., to be used in internal application.

At present, there is still a difference of opinion among the leading advocates of internal fixation in hip fractures as to whether the fracture itself should be exposed by incision and any malposition corrected, or whether the fracture should be nailed blindly after x-ray, anteroposterior and lateral, has proved that it has been correctly reduced.

Cubbins, Callahan and Scuderi, at Cook County Hospital, have a large series of cases in which they have performed open operation with the inspection of the fracture, checking its position and correcting any malalignment, then fixing it with a two-flange nail. Their results have been excellent. Cubbins states that in about one out of seven cases there is interposition of capsule or synovia between the fracture surfaces which would, of course, inhibit union. He gives this as one of his main reasons for resorting to open operation. That this complication occurs in such a high percentage is questionable, for if this were true, the percentage of union after blind nailing would be much lower. Smith-Petersen, who formerly did all of his cases by open operation through a large incision devised by himself is now using the blind nailing method. A complete description of his technique is given in an article recently published.[2]

We believe that the surgeon should be prepared to do either open or blind operation. If, after attempting a reduction of the fracture by manipulation, the check-up x-rays do not show a correct position, it would then be well to expose the fracture by incision, removing any interposed tissue or releasing any fragment that might be

caught in the capsule. The fracture could then be properly reduced and nailed. In the majority of cases, the much sim-

Cannulated coxa protractor.

Steinmann pin in place guided correctly by protractor.

V Mueller & Co Chicago

Driving iron

Fig. 1. This cannulated coxa protractor was devised and perfected through the collaboration of Drs. J. C. Dean, Ivan G. Ellis, J. N. Sisk and J. A. Jackson, and Mr. Larson of the V. Mueller Co.

pler method of blind nailing should be performed.

One of the most difficult problems in the blind nailing method has been to drive the nail at the correct angle of the neck with the shaft. To overcome this difficulty, a cannulated protractor devised by Dean and others and modified by the author insures the correct placing of the nail.

The protractor consists of two quarter segments of a circular metal disc $\frac{1}{16}$ inch thick, with a radius of $1\frac{1}{2}$ inches, each segment graduated in degrees at the margin of the circular edge. These disc segments are fixed to each other in such a way that they exactly coincide, but are spaced apart by small metal lugs. Into the space is inserted a metal cannula, large enough in diameter to take a $\frac{3}{32}$ Steinmann pin,

with one end of the cannula fixed on a freely rotating axis at the center of the arc of the protractor. Over the cannula (and held in any desired position by a set nut threaded on it) is an indicator fashioned to point to the graduations on both faces of the protractor. Near each end of the base of the protractor is a sharp metal tooth $\frac{3}{16}$ inch long for attaching the protractor to the bone. This is accomplished by a small driving iron designed for the purpose. (Fig. 1.)

It is our practice, when a patient with a suspected fracture of the neck of the femur comes to the hospital, to have the case referred directly to the x-ray department, unless the victim is in marked shock. We do this to avoid unnecessary moving and handling of the patient. One-quarter grain of morphine is given and anteroposterior plates of the pelvis, including the upper ends of both femurs, are taken. A lateral view of the neck of the femur on the

injured side is also obtained. Should a fracture be present, the patient is transferred to the fracture operating room where

tion of 20 degrees, and the knee in 90 degree flexion.[3]

At this point, I should like to emphasize

FIG. 2. Special deep retractor.

a bed equipped with a Balkan frame and a Thomas hip splint has previously been made ready. By means of skin traction, about 10 pounds of weight are applied, and the foot of the bed is elevated to provide countertraction. The Thomas hip splint merely acts as a cradle in which to hold the limb and no pressure of the ring against the tuberosity of the ischium is allowed. The patient is now referred to a hospital room and careful study of his general condition is begun, including an immediate urinalysis and blood urea. Since these patients are usually elderly and debilitated, proper measures are at once instituted to fortify them against shock, uremia, etc.

During the ensuing week, the patient is kept as comfortable as possible, and if, at the end of that time, his condition will permit, he is scheduled for operation. He is placed on an ordinary operating room table which has been fitted with a tunneled x-ray film holder. A low procaine spinal anesthesia is given. By the Whitman, Leadbetter, or other manipulation, an attempt is made to reduce the fracture. The opposite limb is flexed at the knee and the foot fastened to the top of a stool at the side of the table. After reduction, an assistant holds the injured limb with abduction of 25 degrees and internal rota-

that there must be perfect teamwork and coöperation between the surgeon and the roentgenologist. A shock proof x-ray unit is indispensable.

The fracture is checked by anteroposterior and lateral x-ray films. If they show a good reduction and moderate valgus position, we are ready to proceed with the operation. The patient is draped and a lateral incision about 5 inches long is made, exposing the trochanter. A specially made, deep retractor with teeth has been of considerable aid in keeping the femur accessible. (Fig. 2.) Approximately ¾ inch below the lower border of the trochanter, in the middle of the shaft, a punch mark is made, which is to be the starting point of a Steinmann pin.

The cannulated protractor, set at the proper angle previously determined from the x-ray film, is now attached to the shaft of the femur with the inner end of the cannula in juxtaposition with the punch mark below the trochanter. A ³⁄₃₂ Steinmann pin is placed in the cannula of the protractor and drilled through the neck and head barely into the acetabulum. By drilling the pin into the acetabulum, the head is steadied and prevented from rotating when the nail is driven in.

Check-up anteroposterior and lateral x-ray films are made and if the pin is

properly placed, as it should be, the protractor is removed and the cannulated Smith-Petersen nail of previously meas-

The incision is closed with a few deep and fascial sutures of light chromic catgut and the skin with skin clips. The Thomas hip

FIG. 3. A, Intracapsular fracture of neck of femur in 84 year old woman (May 12, 1937). Up in chair in one week, on crutches in two weeks, walking without crutches in six weeks with full weight bearing. B, Steinmann pin in place. C, Smith-Petersen nail giving valgus position. D, nail removed in eleven months. Solid bone union.

ured length is driven home and impacted by a special Smith-Petersen tool. In addition, a counterpin is inserted through the head of the Smith-Petersen nail which serves to anchor the pin to the shaft of the femur and prevents its slipping out should the pin become loosened after insertion. The counterpin should be bent to lock it.

splint is reapplied with 5 pounds of weight for traction, merely to relieve muscle strain.

On the following day, the back rest may be raised. By the end of a week, the traction is removed, physical therapy is started, and the patient may sit in a chair. In ten days to two weeks, the use of

crutches is taught; by three weeks the patient is out of the hospital and in from ten weeks to three months, if x-ray films still show good position, the crutches may be discarded for a cane. Follow-up x-rays should be made every three or four months for two years.

A strong word of caution should be added: It is impossible to tell from the x-ray films when good bone union has taken place in this fracture. Do not remove the nail for at least one year and perhaps longer. It apparently does no harm and will keep the fracture in proper alignment for months, even when no union has as yet occurred.

CONCLUSIONS

By a few comparatively simple modifications, the Smith-Petersen method of fixation of fractures of the hip is now a highly advantageous procedure.

The development of the cannulated coxa protractor has provided an accurate means of driving in the nail at the correct angle.

Removal of the nail should be delayed a year or more, and removed then only on special indication.

REFERENCES

1. VENABLE, C. S., STUCK, W. G., and BEACH, A. Effects on bone of the presence of metals; based upon electrolysis; an experimental study. *Ann. Surg.,* 105: 917 (June) 1937.
2. SMITH-PETERSEN, M. N. Internal fixation of femur fractures. *Surg., Gynec. & Obst.,* 64: 287 (Feb.) 1937.
3. CUBBINS, W. Personal communication.
4. DEAN, J. C. Protractor for nailing fractures of neck of femur. *Wisconsin M. J.,* Feb., 1938, p. 115.

Too much emphasis cannot be placed on the adequate roentgenography of fractures of the femoral neck, with careful study and adequate interpretation thereof, both before and after reduction.

From—"A Textbook of Surgery," edited by Frederick Christopher (Saunders).

UNUSUAL COMPLICATIONS OF OSTEOMYELITIS*

MARCUS H. HOBART, M.D., F.A.C.S.   AND   DONALD S. MILLER, M.D.

Assistant Professor of Surgery, Northwestern   Associate Surgeon, Cook County Hospital
Medical School

CHICAGO, ILLINOIS

THE incidence of carcinomatous degeneration of the skin associated with chronic osteomyelitis has recently been called to the attention of the medical profession by the excellent papers of Benedict[1] and Henderson.[2] This association has been seen in twelve of 2,400 cases reviewed by the first author and five of 2,396 cases seen by the latter author at the Mayo Clinic. At the Cook County Hospital this condition has been seen in seven in approximately 400 cases.[13] The second complication of osteomyelitis with which this paper deals is toxic deafness of the eighth nerve as a sequela of the bone infection. Two cases of this nature are herewith reported. No mention will be made of the more common complications of osteomyelitis, such as septicemia, soft tissue infections, arthritis (infective or sympathetic), and the still less frequent complications of secondary hemorrhages, amyloidosis, fractures and nerve involvements.

Although the history of osteomyelitis dates back to paleolithic times, no mention seems to have been made of any complications of this condition, until Marjolin[3] in 1828 associated carcinomatous degeneration of the skin with scarring of burns, but without an osteomyelitic background. The treatment of osteomyelitis, particularly during early wartime periods, by amputation, may have been the cause of infrequent local complications. Wilensky[4] makes no mention of this complication in his comprehensive text on osteomyelitis, and Putti[5] and Haas[6] also have not seen this association occur. Orr[7] does not mention it in a recent communication. This complication, he believes, may be due to frequent daily dressings of the wound. Toxic neuritis of the eighth nerve, although not uncommon in various bodily infections, is a rarity as a sequela of osteomyelitis. This has been verified by Lewy.[8]

One of the earliest reports made was by Dittrich,[9] who in 1847 reported one case with a biopsy done by Rokitansky. Following this, single and multiple case reports were made by various authors until at the turn of the century about thirty of such cases had been reported. Mathieu and Kohn[10] in 1920, Hitzrot[11] in 1921, and Vernengo[12] in 1928 contributed to the literature with several cases. In the more recent reports, Benedict,[1] cites a case which revealed a chronic draining sinus for only one year, although trauma preceded the malignant foot condition for several years. We, likewise, have two such cases with relatively short draining periods.

The seven cases that are herewith reported show the changes seen in other reports: the two types of cancer seen were the exuberant granulation type with heaped up nodular areas around the source of drainage (Fig. 1), and the deep crateriform type with little local reaction. (Fig. 2.) Both give histories of chronic discharge with neglected or inadequate treatment, foul odor, and hemorrhages from invasion of the malignant process into the soft tissues. Patients seen in a charity hospital such as the Cook County are usually the indigent poor, unable to have the proper medical attention. After years of chronic drainage and mild symptoms referable to their lesion they come to the clinic for treatment.

The two cases of eighth nerve deafness occurred in boys of school age. It is inter-

esting to note that there was a definite period of neglected treatment between the onset of the acute symptoms and the primary operative drainage.

forty years. Two patients definitely gave a short history of chronic drainage for a one and one-half and a two and three-quarter year period. The latter patient,

CHART I
UNUSUAL COMPLICATIONS OF OSTEOMYELITIS
Malignancies of Skin Following Osteomyclitis

Name	Age	Occupation	Duration Osteomyelitis	Symptoms and Findings	Complications	Results—Sequelae
S. C.	56	Laborer	1½ ycars.	Pain, swelling, mass locally (prolif.), anemia.	Carcinoma skin at lower third thigh.	Amputation—result good. 4-4-1936.
J. S.	56	Laborer	50 ycars.	Mass locally (prolif.), foul odor, anemia.	Carcinoma skin at lower third leg.	Amputation—result good. 7-1-35.
M. K.	73	Housewife	40 ycars.	Mass locally (prolif.), foul odor, hemorrhage, anemia.	Carcinoma skin at heel.	Death from sepsis— (before surgery)
F. M.	68	Laborer	2 ycars, 9 mo.	Pain, foul odor, hemorrhage (lx), mass (prolif.)	Carcinoma of skin at middle third of leg.	Amputation—result good. 7-8-36.
W. O.	52	Clerk	40 years.	Mass locally (prolif.), hemorrhage 6x, foul odor, anemia.	Carcinoma of skin at ankle arca.	Amputation—result good. 1-29-37.
W. R.	53	Laborer	43 years.	Mass locally (prolif.), pain and discharge.	Carcinoma of skin at mid-third of leg.	Extensive curettement —result good.
Dr. L.	62	Physician	45 years.	Mass locally (prolif.), and chronic drainage.	Osteogenic sarcoma at the lower third of leg.	Amputation—result good. 6-1-36.

Toxic Neuritis Eighth Nerve Following Osteomyelitis

Name	Age	Occupation	Duration Osteomyelitis	Symptoms and Findings	Complications	Results
A. O.	16	School	1 year before surgery.	Pain, discharge.	Toxic neuritis eighth nerve. Bilateral.	Complete nerve deafness; amyloidosis.
E. L.	16	School	5 mo. before surgery.	Pain, discharge.	Toxic neuritis eighth nerve.	Complete nerve deafness; death from brain abscess.

Age of Patients and Duration of Symptoms. The age of the patients in the cancer group varied from 52 to 73 years. (Chart i.) The two patients developing toxic neuritis of the eighth nerve were 15 and 16 years old respectively. The former group gave a history of drainage periods of from one to

however, had a chronic bone infection for many years before. The third patient gave a history of drainage for fifty years, the fourth a history of forty years' duration, the fifth also 40 years of drainage, the sixth thirty-seven years, and the seventh patient a history of forty-five years drainage.

The patient most recently seen was operated upon by Dr. Philip Lewin; the history showed a draining sinus for forty-five

vaseline pack, and suffered no recurrence locally. In one case septicemia and death occurred before any treatment was insti-
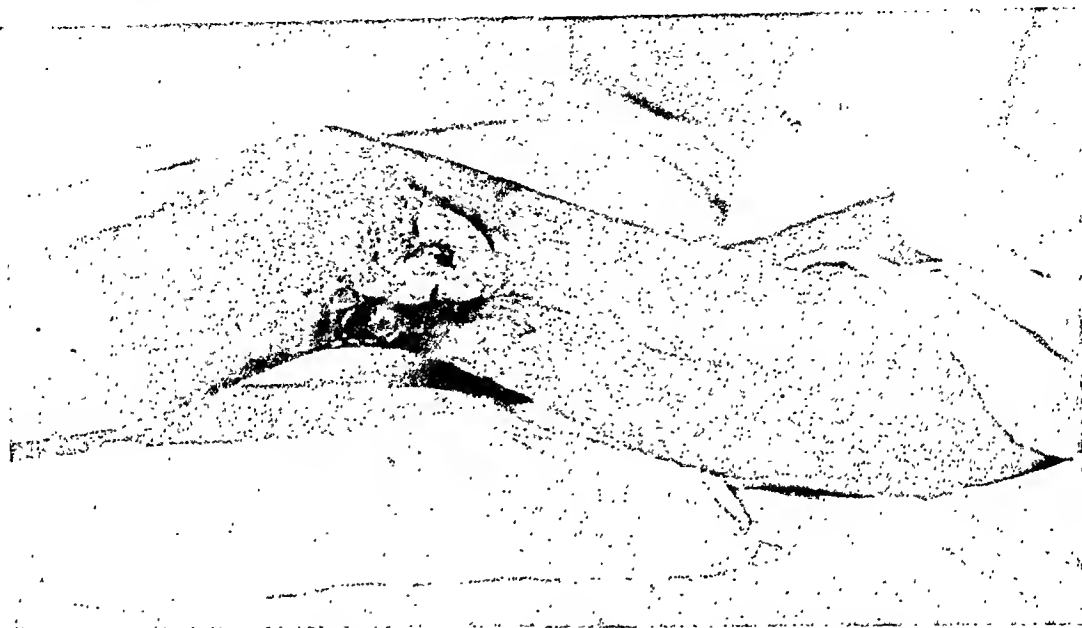
FIG. 1. Case I, showing the exuberant granulation type of skin malignancy.

years. The biopsy by Dr. Jaffe revealed an osteogenic sarcoma. This case will be described in a subsequent paper.

Occupation. The occupations of these patients were: laborers (four), one clerk, one housewife, and one physician. The two who developed toxic neuritis of the eighth nerve were schoolboys.

Location of Lesion and Symptoms. The malignant conditions of the skin in this series were all in the lower extremities. Two were in the ankle area, four in the leg, and one occurred in the thigh. The symptoms predominantly were those of chronic discharge over many years (except in one case), slight pain locally, foul odor and hemorrhages in three cases. Four of the seven cases had enlarged inguinal lymph glands.

Treatment. In five of the seven cases amputation was done. For those malignancies occurring in the leg, amputation was performed below the knee; for the single instance at the lower third of the thigh, an amputation at the mid-third of the thigh was done. One of the patients had a very wide currettement, with an Orr

tuted. This was in a very old feeble lady who also suffered from nephritis and organic heart disease. All of the cases were proved by biopsy.

FIG. 2. Case V. Osteomyelitic ulcer with carcinomatous edge. Deep crateriform type.

Course. As this paper is written, follow-up includes periods of from one to three years. There has been no recurrence nor metastasis of any type. In one patient it was necessary to do three biopsies before

a microscopic diagnosis could be made. The patient operated upon by Dr. Berkheiser, where a wide curettement was is slight or nil. The patient disregards the slight increase in symptoms and just "drops in" to the clinic for a checkup.

Fig. 3 Case IV. X-ray showing osteomyelitis of middle third of tibia with marked bony defect.

performed, is doing exceptionally well. The two cases of toxic deafness of the eighth nerve definitely show that this condition arose during the neglected treatment of the bone infection, both cases having had no surgical intervention at the time the acute symptoms arose.

X-Ray Appearance. Although the x-rays were not characteristic of soft tissue malignant invasion, there were some suspicious moth-eaten areas with rarefaction. (Fig. 3.) One should be on the lookout for these when the clinical history is suggestive. Bone proliferation here was at a minimum, and the eroded areas in the bone corresponded to the soft tissue ulcers. (Fig. 2). There was considerable decalcification of the entire bone.

SUMMARY

The fact that these malignancies are usually engrafted on an old ulcer, does little to alarm the patient already inured to his osteomyelitis. Usually the disability here

The findings of chronic discharge, foul odor, pains of a mild nature, the clinical appearance of the lesion and a biopsy report furnish evidence enough that surgery must be instituted. The two cases showing the relatively short drainage period, should impress one with the probability that a low grade osteomyelitis may exist for many years, with an exciting factor such as trauma or infection as the inciting cause. It is hardly conceivable that lowered skin resistance would be a predisposing factor in the malignancies, since perfectly normal skin was present before any clinical appearance of the lesions. It is quite possible that in the other five cases the continued irritation, and poor skin resistance would lead to infection and malignancy. The patients who developed the toxic neuritis show a latent period between the acute onset of the osteomyelitis and the primary surgical intervention during which deafness started, continued and was not arrested nor helped by the treatment of the osteo-

myelitis. The treatment of skin malignancies by amputation or by wide curettement of the involved parts insures a good result.

times. No history of onset was given. Because of the extremely serious condition of the patient only a biopsy was taken. This revealed a very anaplastic hornifying squamous cell carcinoma.

Fig. 4. Case 1. Microscopic section showing carcinoma cells.

The first of the toxic neuritis patients developed a brain abscess and succumbed; the second is still living with a complicating amyloidotic condition.

CASE REPORTS

1. S. Z., white male of 56, came into the clinic with the history that he had an open, draining area of the left thigh for a year and a half. Because of swelling and tenderness of the left knee a physician advised surgery. At this time he was operated upon. Drainage continued. The patient came to the clinic because of the persistence of drainage. At this time malignancy was disclosed. (Fig. 4.)

2. J. S., white male of 56, had had an acute attack of osteomyelitis at the age of 6. An incision was then made and continued to drain on and off for this period of fifty years, exuding bone and blood for some years. Biopsy report revealed malignancy of a squamous type. (Fig. 5.)

3. M. K., white female of 73, entered the surgical ward with a history of having had open draining areas of the heel and knee for forty years. These lesions opened and closed many

4. F. M., white male of 68, entered the medical ward because of an open lesion of the leg. The history given was that two and one-half years before, a small papule started locally and continued to grow. Several months prior to this, the patient was in an automobile accident and traumatized his leg and thigh. The lesion continued to get larger, and when he presented himself in the clinic the patient had a typical osteomyelitic lesion with skin changes. Biopsy revealed hornifying epidermoid carcinoma.

5. W. O., male, age 52, entered the orthopedic clinic with the complaint that for forty years he had had a chronic draining area in his ankle. This followed jumping to the ground from a barn. He was operated upon for acute osteomyelitis. Since then he has had several curettements, but the deep lesion persisted. Biopsy of the edge of the lesion revealed hornifying squamous cell carcinoma.

6. W. R., male, aged 53, came to the Rush Medical Dispensary on Dr. Berkheiser's service. The history obtained at that time disclosed that at the age of 10 he was run over, sustaining a compound fracture of the leg. The open area had been draining since. Several months before his admission to the clinic, the

wound was closed, but reopened. Biopsy revealed (after second attempt) squamous cell carcinoma.

bone. He was operated upon, made an uneventful recovery, but deafness continued. In May, 1937, he developed symptoms of brain abscess
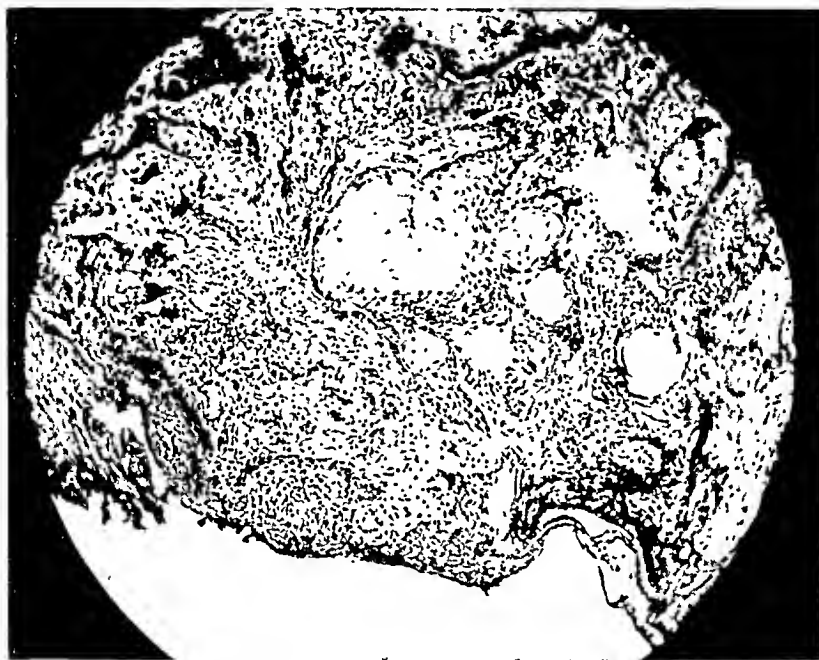
FIG. 5. Case II. Photomicrograph showing squamous cell carcinoma.

7. Dr. L.., male, aged 62, reported that forty-five years before he had developed an osteomyelitis of the leg. This was curetted ten years later and remained closed for some time, but later reopened and continued to drain in spite of several closures. Several months before admittance to the hospital he noticed a nodular swelling in his leg, and then came to the hospital where a sarcoma was diagnosed.

8. A. O., male, aged 15, was admitted to the Cook County Hospital because of an old chronic osteomyelitis. At this time he complained of roaring in his ears and an inability to hear well. His history was that of an acute onset of pains in the leg, associated with fever which was treated with hot packs and rest. Surgery was instituted several months later and an osteomyelitis was disclosed. Deafness was slow and progressive. At present this patient is totally deaf, and has developed an amyloid condition.

9. E. L., male, age 16, stated that in April, 1934 he had complained of pain and swelling in the leg. Six months prior to this he had suffered occasional leg pain, which was worse at night. The condition was treated with dry heat and medicines. Deafness came on gradually and x-ray revealed an osteomyelitic condition of the

and succumbed to this condition after twelve days.

CONCLUSIONS

1. Chronic draining osteomyelitis should be carefully watched for malignant changes of the skin.

2. If these cases are treated by amputation or wide curettement, good results are assured.

3. There is a definite relationship between the onset of acute osteomyelitis and the period of treatment; in two cases of neglected treatment nerve deafness occurred.

4. No metastases have occurred to date following amputations.

5. Biopsies should be adequate and repeated, if suspicious areas are seen microscopically.

REFERENCES

1. BENEDICT, E. H. Carcinoma in osteomyelitis. Surg., Gynec. & Obst., 53: 1, 1931.
2. HENDERSON, M., and SWART, H. A. Chronic osteomyelitis associated with malignancy. J. Bone & Joint Surg., 18: 56–60 (Jan.) 1936.

3. Marjolin. *Dict. de med.*, vol. 21, 1828.

4. Wilensky, A. O. Osteomyelitis, its pathogenesis, symptomatology and treatment. New York, 1934. Macmillan.

5. Putti, V. Personal communication.

6. Haas, J. Personal communication.

7. Orr, H. Winnett. Personal communication.

8. Levy, A. Personal communication.

9. Dittrich, L. *Prag. Vierteljabrsscbr.*, 2: 161, 1847.

10. Mathieu and Kohn, T. Epitélioma pavimenteux développé au contact d'un foyer ancien d'oxtéo-myélite chronique. *Bull. et mém. Soc. anat. de Par.*, 90: 210, 1920.

11. Hitzrot, J. M. Epithelioma in sinus of old osteomyelitis. *Ann. Surg.*, 73: 247, 1921.

12. Vernengo, M. J. Epitelioma des arrollado en una fistula por osteomielitis cronica. *Bol. y trab. Soc. de cirug. de Buenos Aires*, 12: 801, 1928; *Rev. de cir.*, 7: 557, 1928.

13. Hobart, M. H., and Miller, D. S. Osteomyelitis at Cook County Hospital. *J. A. M. A.*, 107: 1118–1121 (Oct. 3), 1936.

It is easier to fit an artificial limb on the leg with the amputation at the junction of the upper and middle third (or slightly lower), than with an amputation in the lower third. When the amputation is performed in the lower leg, the fibula must be removed at a higher level than the tibia.

From—"Textbook of General Surgery" by Warren H. Cole and Robert Elman (D. Appleton-Century).

HUMAN BITE INFECTIONS*

Paul E. McMaster, m.d.

Assistant Professor of Orthopedic Surgery, University of Southern California Medical School

LOS ANGELES, CALIFORNIA

THE importance of early radical treatment of wounds caused by human bites and the lacerations to hands and knuckles received in fist fights is often overlooked or not realized. Such injuries frequently cause prolonged infections which may lead to abscess and extensive soft tissue necrosis, suppurative tendonitis, periostitis, osteomyelitis or septic arthritis, or all of these. Some of these infections may become so severe that amputation is necessary, and they may even terminate fatally. It is not uncommon to see stiff and useless fingers from ankylosis of joints or destruction of tendons. Sixty-eight cases of human bite infections have been studied in the past three years and are reported.

A few reports of human bite infections have appeared in the literature. The first such report appears to be the one by Hultgen[1] in 1910. He reported the case of a 7 year old girl who developed an ill-smelling, fetid infection of a finger caused by biting her finger nails. Smears of the discharge showed the fusiform bacillus and the spirocheta denticola. The following year Peters[2] reported five cases of infection due to Bacillus fusiformis, two of which were of the hands following fist fights. One of the latter cases showed both the fusiform bacillus and spirochete of Vincent, while the other yielded only the fusiform bacillus. Peters noted the occurrence in these two cases of intense edema, swelling, and foul discharge. Hennessy, Madras, and Fletcher[3] reported a case of a thumb bite in which the bacillus-spirillum combination of Vincent was shown by smear. The infection became so severe that amputation of thumb was necessary. Flick[4] reported the case of a severe infection following a bite on the thumb. He advised amputation which the patient refused. Death occurred sixteen days after the bite.

Other articles dealing with this condition are those by Pilot and Meyer,[5] Fuller and Cottrell,[6] Owen,[7] Mason and Koch,[8] Bates[9] and Welch.[10]

Occurrence and Location of Lesions. The most common type of injury from human teeth predisposing to serious infection is that received in bare fist fights. These lacerations involve usually the second, third, and fifth metacarpophalangeal regions. Thirty-eight of this series of sixty-eight cases followed fight injuries, while thirty were wounds directly inflicted by one human biting another. Most of the bites occurred on fingers and hands, while some involved the forearm, axilla, and thigh.

Thirteen of the thirty bite cases were of women, while the remaining seventeen bite cases, and all of the fight cases were of men. Ages ranged from a few months to seventy-two years.

Course of Infection. Few of the patients in this series were seen early. The usual story which a patient gave on admission was that he had been bitten or had received a laceration of his hand in a fight from two to four or more days previously. The wound had been treated either at home by the patient or it had been dressed or sutured by a physician. Pain and swelling had subsequently developed within twenty-four to forty-eight hours.

Examination of these wounds, which were a few days old, revealed redness, swelling, increased local heat and tenderness. Lymphangitis and axillary adenitis were observed in several cases. Limitation

of finger motion due to pain and swelling was not uncommon. In some cases a seropurulent, foul-smelling discharge from being open and superficial, allowed adequate drainage. All such cases were treated with oxidizing solutions such as hydrogen
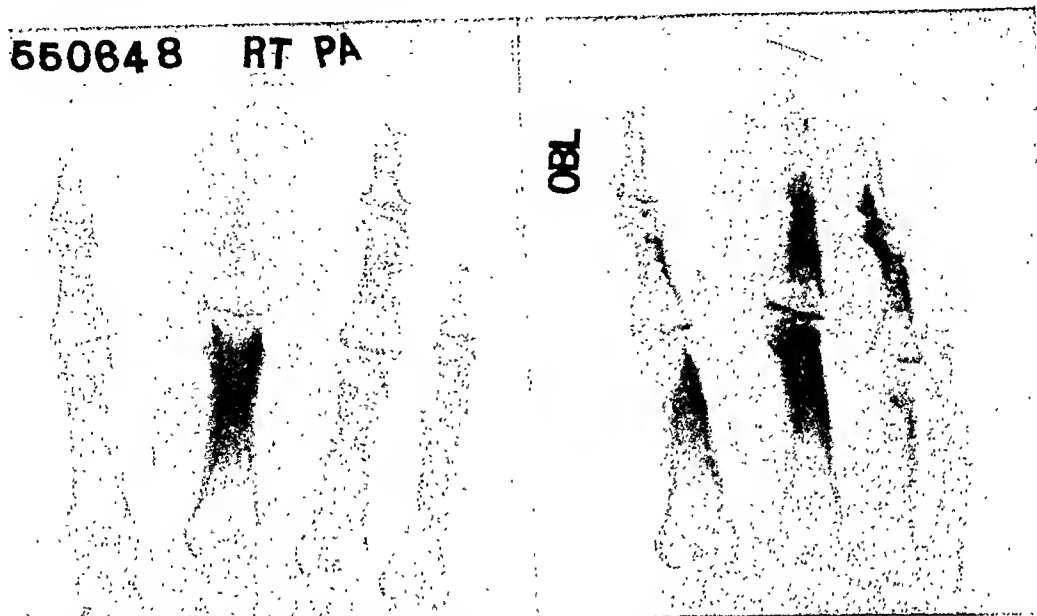
Fig. 1. Roentgenograms showing destructive arthritis of distal interphalangeal joint of middle finger with associated osteomyelitis, periostitis and diffuse soft tissue swelling, which were present on entry three weeks after bite. Smear revealed Vincent's organisms. Finger was amputated and patient made an uneventful recovery.

wounds had developed. A few of the patients appeared quite ill and the temperature readings varied from 99°F. to 102°F. In those patients not seen for two weeks or more after the injury, extensive cellulitis, lymphangitis, regional adenitis, local tissue necrosis, suppuration, and a very offensive, malodorous discharge were usually present.

Treatment. The cases which were seen within the first few hours after injury (there were only three, of which two were finger bites and one a forearm bite) were treated by cauterization. Phenol was used on two and nitric acid on the other and the wounds left open. The dressings were kept moist with hydrogen peroxide. All healed without complication.

The avulsive bite cases in which finger ends, or segments of skin were completely bitten away, as well as the superficial abrasions, did not offer difficulty in treatment, and complications, beyond a temporary mild local inflammation, did not occur. These wounds had bled freely and,

peroxide or sodium perborate and left open. In one case, a finger end, which was almost but not completely bitten off, had been replaced and sutured before entry. A subsequent severe felon-like infection with osteomyelitis of the distal phalanx resulted. After several weeks of treatment, healing finally occurred.

The severely infected wounds, which were seen several days or more after injury and in which there was much swelling and redness, were opened widely (all stitches being removed when present) and necrotic tissue excised. Severe infections occasionally followed penetrating bite wounds but most commonly the worst infections developed in the lacerations by teeth from fist fights at the metacarpophalangeal joint regions. After excision of the necrotic tissue all wounds were left open to drain and a majority were then compressed with oxidizing agents such as hydrogen peroxide soaks or sodium perborate powder sprinkled in the wounds and then covered with warm moist aqueous dressings. The others

were treated with continuous hot wet dressings of magnesium sulfate or boric acid. Those patients treated with oxidizing agents appeared to heal more rapidly than those treated with boric or magnesium sulfate packs.

Despite the fact that these wounds were incised widely, necrotic tissue excised, and the wounds left wide open to drain, thirty-one of the sixty-eight patients had to be hospitalized for two weeks or more. Some were in the hospital for several weeks and one for more than twenty-nine weeks.

Bates reported successful treatment without serious complications in over one hundred cases treated by radical electro-cautery excision. He used it not only in early cases but in late cases in which there was much redness and swelling. Judging from his reported results and the success in the few cases of this series treated by acid cauterization, it would appear that some form of cautery treatment for these cases might be the preferable method.

Intravenous neoarsphenamine was tried several times in this series, both alone and in combination with local treatment. No noticeable influence on the course of the infection was noted.

Bacteriology. Smears and cultures were made of the discharge in forty-one cases. Ten smears showed both the fusiform bacillus and the spirochete of Vincent, while eleven yielded only the fusiform bacillus. The other twenty cases revealed one or a combination of the following: pus cells, diphtheroids, staphylococci, strepto-cocci, and cellular débris. Five cultures were negative; however, the majority yielded staphylococcus aureus and a few showed Streptococcus viridans and Strep-tococcus hemolyticus. Blood cultures in a few of the severe cases with high tempera-ture readings yielded no bacterial growth.

Complications. Complications of vari-ous types and degrees occurred, depending on the severity of the initial lesion and the elapsed time before adequate treatment was given. Those following the bite infec-tions were less severe as a rule than those

occurring after fist fight infections. Exten-sive soft tissue necrosis resulted in one forearm and one thigh after bite lacerations to these parts. Bites to fingers led to periostitis, suppurative arthritis, and osteo-myelitis of phalanges in a number of cases. (Fig. 1.)

Severe complications followed the fight wounds. These injuries, which involve usually the second and third metacarpo-phalangeal joint regions, perforate the skin and extend into the periarticular or intra-articular spaces. As the closed fingers (fist) are extended, the dorsal skin overlying the knuckles tends to retract, thus sealing over the deeper wound and establishing anae-robic conditions · for the human mouth organisms. The course of the infection from these wounds has been well shown in the experimental studies on cadavers by Mason and Koch. Complications which followed these wounds were cellulitis, lymphangitis, abscess, and necrosis of extensor tendons. Suppurative arthritis was not uncommon and in seventeen cases was associated with not only destruction of articular cartilage but osteomyelitis of adjacent phalangeal and metacarpal bones as well. (Figs. 2 and 3.) Palmar space abscess with necrosis and sloughing of flexor tendons to fingers occurred in a few cases from extension of the dorsal infection either across the cap-sular space, or via the pericapsular tissues, or along the lumbrical muscles.

Amputations were necessary in nine cases. Two were of fingers which had become stiff and useless after the infections had cleared up. The other seven were done because of extensive soft tissue and bone necrosis. Four of these fingers were re-moved at the metacarpophalangeal joints and three included in addition the respec-tive metacarpal heads. One in the former and one in the latter group continued with spreading severe infections of hand requir-ing amputation through the forearm.

Pathologic Studies. Gross examination of the amputated specimens revealed skin ulceration, subcutaneous abscess and ex-tensive soft tissue necrosis, including in

some cases destruction of extensor and flexor tendons. Intra-articular pus with necrosis of articular cartilage and a moth-

FIG. 2. Roentgenograms showing destructive arthritis of third metacarpophalangeal joint with periostitis of adjacent phalanx and metacarpal, also considerable diffuse soft tissue swelling. These changes were present one month after a fight injury. Smears of the draining wound yielded fusiform bacilli. Amputation was required.

eaten appearance of adjacent bone was seen. The microscopic sections of soft tissue showed heavy, diffuse infiltrations of polymorphonuclear leucocytes and in some areas abscess. Sections of bone showed necrosis of articular cartilage and diffuse infiltrations of polymorphonuclear leucocytes in the cancellous spaces with bone destruction taking place by mononuclear and giant cell osteoclasts.

Comment. It is difficult to estimate the frequency of human bite infections. They probably are not uncommon, for, although most writers report only one or a few cases, Bates obtained records of over 200 cases from three Philadelphia dispensaries during a seven year period. Welch reported a series of eighteen cases and Mason and Koch thirteen. In this present series, sixty-eight cases are reported in a three year period. A number of these patients, apparently ashamed of their injuries, misstated the cause and only after careful questioning did

they reluctantly admit the truth. No doubt many minor abrasions caused by teeth wounds progress to uneventful recovery. Many others of a more serious nature are likely not properly diagnosed, due to misstatements by the patients or insufficient medical study.

The severity of these infections is seemingly due to the Vincent's organisms implanted deeply in the tissues under anaerobic conditions. Although the bacillus-spirillum combination was demonstrated in only ten of forty-one smears and fusiform bacilli in eleven others, it is probable that a higher percentage of cases harbored the organisms deep in the tissues surrounding the wound or these organisms may have been destroyed along with the tissue necrosis.

Vincent's organisms are facultative anaerobes. As a large number of human mouths harbor them (smears taken from a hundred mouths in the Dental Clinic of the Los Angeles County Hospital revealed that seventy-eight patients had both the bacillus and the spirochete), wounds caused by

human teeth should not be primarily closed. Observations of all such wounds in which primary closure by suture had been done showed suppuration and necrosis.

involved the metacarpophalangeal joint regions, having resulted from fist fight injuries. Thirty were caused by actual bites.
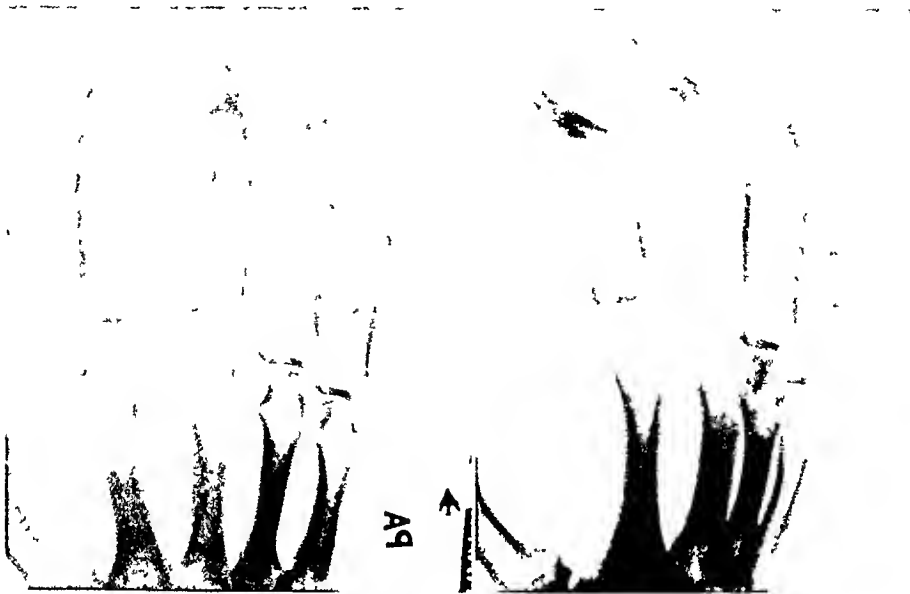
FIG. 3. Roentgenograms showing destructive arthritis of middle metacarpophalangeal joint with associated osteomyelitis and some periostitis six weeks following fight injury. Segments of both extensor and flexor tendons of the finger sloughed out. A stiff useless finger resulted.

Complications of a serious and prolonged nature are likely to occur, unless early adequate treatment is given. Avulsive bites and superficial abrasions should be cleansed and left open. Application of oxidizing agents will usually suffice to inhibit growth of the Vincent's organisms. Should the bite be by an unknown assailant or one with poor mouth hygiene, cauterization of the wound is a safer procedure. All penetrating wounds should be excised completely and the wounds left open. Excision by cautery knife appears to be preferable to "cold steel." The cases seen late with cellulitis, abscess, and necrosis require incision and drainage with removal of necrotic tissue. This too may be done with a cautery knife. More serious complications, such as those necessitating amputation, must be individualized.

SUMMARY

1. Sixty-eight cases of infections caused by human teeth are reported. Thirty-eight

2. Superficial abrasions and avulsive bites did not cause serious infections. Penetrating bites and fist fight injuries to knuckles resulted in the most serious complications, of which the most common were abscess, tissue necrosis, sloughing of tendons, osteomyelitis, and suppurative arthritis. The discharge in these cases was very offensive and malodorous. Late sequelae were stiff fingers from tendon destruction, soft tissue scarring and ankylosis of joints.

3. Nine amputations were done. Two of these were of stiff, useless fingers after the infection had subsided. Seven were of fingers because of extensive severe active infection, and of these two later required forearm amputations to control spreading infections.

4. Smears of discharge in forty-one cases revealed both the fusiform bacillus and the spirochete of Vincent in ten, while eleven others showed the fusiform bacillus but not the spirochete. A majority of the cultures

from these cases yielded Staphylococcus aureus and a few yielded Streptococcus viridans and Streptococcus hemolyticus.

5. Oxidizing agents, such as hydrogen peroxide and sodium perborate, applied to the wounds appeared to produce more rapid healing than when ordinary hot wet soaks were used. Intravenous neoarsphenamine had no appreciable influence on the repair of these wounds.

CONCLUSIONS

1. Superficial abrasions and avulsive bites, if left open, will as a rule heal without serious complications. Cauterization is a safeguard and applications of oxidizing agents is advisable.

2. Penetrating wounds from human teeth should be widely excised early and cautery excision is probably preferable.

3. Late severe infections should be treated by wide incision and excision of necrotic tissue.

4. All wounds caused by human teeth should be left open for drainage and not primarily closed.

REFERENCES

1. Hultgen, J. F. Partial gangrene of the left index finger. *J. A. M. A.*, 55: 857, 1910.
2. Peters, W. H. Hand infection apparently due to Bacillus fusiformis. *J. Infect. Dis.*, 8: 455, 1911.
3. Hennessy, P. H., Madras, C. M., and Fletcher, W. Infection with the organisms of Vincent's angina following man-bite. *Lancet*, 2: 127, 1920.
4. Flick, J. B. Gangrenous infection of the hand and forearm following human-bite. *Ann. Surg.*, 90: 450, 1929.
5. Pilot, I., and Meyer, K. A. Fusiform bacilli and spirochetes. xii. Occurrence in gangrenous lesions of fingers: Report of a case. *Arch. Dermat. & Syph.*, 12: 837, 1925.
6. Fuller, C. R., and Cottrell, J. C. Infection with organisms of Vincent's angina following human bite. *J. A. M. A.*, 92: 2017, 1929.
7. Owen, H. R. Chancre complicating laceration of hand. *Ann. Surg.*, 87: 783, 1928.
8. Mason, M. L., and Koch, S. L. Human bite infections of the hand, with a study of the routes of extension of infection from the dorsum of the hand. *Surg., Gynec., & Obst.*, 51: 591, 1930.
9. Bates, W. Electrocauterization in the treatment of human bites. *Ann. Surg.*, 93: 641, 1931.
10. Welch, C. E. Human bite infections of the hand. *New England Med.*, 215: 901, 1936.

A CLINICAL PATHOLOGIC CLASSIFICATION OF ACUTE APPENDICITIS AND PERITONITIS COMPLICATING PERFORATIVE APPENDICITIS*

JOHN O. BOWER, M.D.

Clinical Professor of Surgical Research, Temple University School of Medicine

PHILADELPHIA, PENNSYLVANIA

THE following conclusions deduced from observations made during the course of clinical and experimental investigations on acute appendicitis and spreading peritonitis complicating perforative appendicitis prompted this presentation:

One in every three patients admitted to the hospitals in the United States with a diagnosis of acute appendicitis has an associated peritonitis.

The operative mortality of spreading peritonitis complicating acute perforative appendicitis is excessively high.

A further study of the morbid and microscopic tissue changes that occur in appendicitis will not aid physicians or surgeons in solving the problem of the high mortality.

The importance of the clinical pathologist as a consultant in the proper management of patients suffering with an acute abdomen is not realized by physicians or surgeons.

The study of normal and abnormal processes in the living and dead has, as its ultimate aim, the understanding of disease and its prevention or cure. Pathologists, by persistent and painstaking studies of gross and microscopic changes, have enabled physicians and surgeons to make earlier diagnoses by correlating tissue or cell changes with the clinical manifestations of the disease. This has been true particularly of subacute and chronic lesions. Improved surgical management of acute progressive processes, however, has been made possible mainly through clinical and pathologic investigations, and not through the examination of tissues removed at the operating or post-mortem table. There are two reasons for this:

1. Acute progressive lesions are usually bacterial in origin and immediate surgical intervention may not be indicated; it may even prove fatal if carried out. Microscopic examination of the earliest cell changes, therefore, is impossible.

2. In the acute infections requiring surgery, early cell changes in the organs or tissues primarily involved are not in themselves sufficient to produce death. This precludes the possibility of post-mortem examination of them.

The above is true of acute lesions involving many of the abdominal organs but particularly true of acute infections of the appendix. Little has been added to our knowledge of the gross and the microscopic changes in acute lesions of the appendix during the past twenty-five years. The following pathologic classification is taken from John B. Deaver's book on appendicitis written in 1913:[1]

Pathology of Acute Appendicitis:
  (1) Catarrhal
  (2) Interstitial
  (3) Ulcerative
      (a) Nonperforative
      (b) Perforative
  (4). Suppurative
  (5) Gangrenous.

Since Deaver's classification, pathologists have added appendicosis, characterized by degenerative rather than inflammatory changes in the mucous and submucous

coats. During the past forty years in the United States alone, approximately fifteen million acute appendices have been removed and literally millions of them have been examined microscopically. Further a patient lives or dies is seldom determined by pathologic changes in the tissues beneath the serosa or the nature of the contents of the lumen of the appendix. When the serous membrane is intact, one

CLINICAL PATHOLOGIC CLASSIFICATION OF APPENDICITIS AND PERITONITIS

Classification	Pathology	Immunologic Response	Result
1. Localized lesion (a) Acute catarrhal...... (b) Acute suppurative... (c) Acute gangrenous.... (d) Acute gangrenosuppurative..........	Serous coat intact.	Approximately 20 per cent.	1 in 183 dies.
2. Localizing process........	Serous coat ruptured but sealed with fibrinous exudate, omentum, visceral or parietal peritoneum.	If undisturbed, local tissue immunity develops and an abscess forms. If operated upon, spreading peritonitis is frequently induced.	Dependent upon management.
3. Localized abscess........	Abscess.	Local and general immunity develops after sixth or seventh day.	1 in 44 dies.
4. Spreading peritonitis.....	Spreading process.	Dependent upon management and the individual resistance.	1 in 4 dies.

study might reveal earlier cellular or possibly intracellular changes which would affect our concept of the histogenesis of the disease, but it would not give the internist or the surgeon the necessary data with which to make a more accurate diagnosis of a pre-perforative or early perforative appendicitis, nor would it materially affect the mortality of this disease.

The clinical pathologic classification emphasizes:

1. Pathology in the living.

2. The important rôle immunity—local tissue and general—plays in the management of spreading peritonitis.

3. The correlation of symptoms, signs and gross changes found at operation with what happens to the patient.

1. *Pathology of the Living.* Nature has been generous in allowing surgeons great latitude of judgment without exacting compensation in the recognition of pre-perforative appendiceal pathology at operation. Low or high mortality is dependent upon an intact or ruptured serosa; whether

in 183 dies; when it is ruptured, one in eight dies.

Patients rarely die from appendicitis. Autopsies are performed on patients who die from peritonitis, not appendicitis.

TABLE I
PATHOLOGY AND MORTALITY

	No. of Cases	No. of Deaths	Per Cent Mortality
Acute nonperforative appendicitis 65.6 per cent of cases	12,259	67 1 in 183 dies	0.54
Acute appendicitis and peritonitis 34.4 per cent of cases	6,428	782 1 in 8 dies	12.2

The pathologist cannot aid by the study of morbid changes observed at postmortem. His gross and microscopic findings can only be correlated with the symptoms and signs preceding death. Furthermore,

the post-mortem in many instances is delayed several hours, during which time migration and multiplication of anaerobic and putrefactive micro-organisms produce tissue changes which make an accurate interpretation of preagonal pathology impossible.

Table II shows the number and percentages of those who die from acute appendicitis—the *localized lesion;* of those who die from local peritonitis—the *appendiceal*

TABLE II

PATHOLOGY AND MORTALITY

	Total No. of Cases	Incidence, Per Cent	No. of Deaths	Per Cent Mortality
Acute nonperforative...	12,259	65.60	67	0.54
Acute local peritonitis..	3,855	20.63	88	2.28
Acute spreading peritonitis.....	2,573	13.77	694	28.97
	18,687	100.00	849	4.54

abscess; and of those who die from *spreading peritonitis.* This table does not show the number or percentage of those dying because of the conversion of a localized mass or a localizing process into a spreading one. The percentage so converted can be estimated with a fair degree of accuracy.

In sixty-six out of any given hundred acute appendices the serous coat is intact; in thirty-four cases the peritoneal coat has been ruptured. (Table I.) Approximately fourteen of these thirty-four cases belong to the spreading peritonitis group. (Table II.) Of the remaining twenty cases, in approximately twelve the surgeon will palpate a definite mass, a localized abscess. This leaves eight cases in every hundred where at operation, one might encounter an appendix in which the peritoneal coat has been ruptured, but leakage has been prevented by plastic exudate, omentum or intestine. (Fig. 1.) This is the *localizing process,* and because of its gradual development, gross tissue changes are not easily recognized.

If the incision has been made over or near the lesion, the preperitoneal tissues are edematous and the parietal peritoneum will be injected; the degree of hyperemia of the visceral peritoneum and the presence or absence of adhesions will depend upon the time that has elapsed between perforation and operation and the resistance of the patient. Free fluid, if present, is usually moderate in amount, cloudy, seropurulent and slightly odorous. A smear may show micro-organisms; cultures frequently do. If rupture occurs in an appendix which has previously been involved in an infectious process, the accompanying appendiceal fibrosis and peri-appendiceal adhesions diminish absorption, and hyperemia of the peritoneum and transudation of fluids will be comparatively less. In a small percentage of cases in this group, the appendix perforates into its mesentery; it is partly or completely hidden by omentum, ileum or caecum and when exposed the capillaries are distended, the peritoneal coat is thickened and edematous and the mesentery is of normal color but markedly thickened at the site of perforation and the free border is usually intimately attached to the cecum or ileum. When the mesentery is caught with a hemostat, seropurulent material will escape if the tip penetrates the involved portion. Perhaps the most important pathologic finding which suggests the concealed perforation is the marked thickening of the omentum caused by the increased vascularity, edema, neutrophilic and clasmatocytic infiltration. The foregoing is all that can be seen through a transverse or muscle-splitting incision. Because of lack of visibility, early circulatory changes in the parietal or visceral peritoneum, filmlike adhesions, peritoneal exudate or indefinitely formed fibrinous plaques may be overlooked, or, in the attempt to locate the appendix with the finger or quarantine the diseased organ with gauze sponges, they may have been covered or obliterated.

In describing the localizing process, the surgeon, of necessity, must draw on his

memory and imagination to complete the picture; the description of details therefore, is not accurate. Unfortunately, in a great

ing one—that while the patient, before operation, was absorbing a gradually diminishing amount of antigen, during the
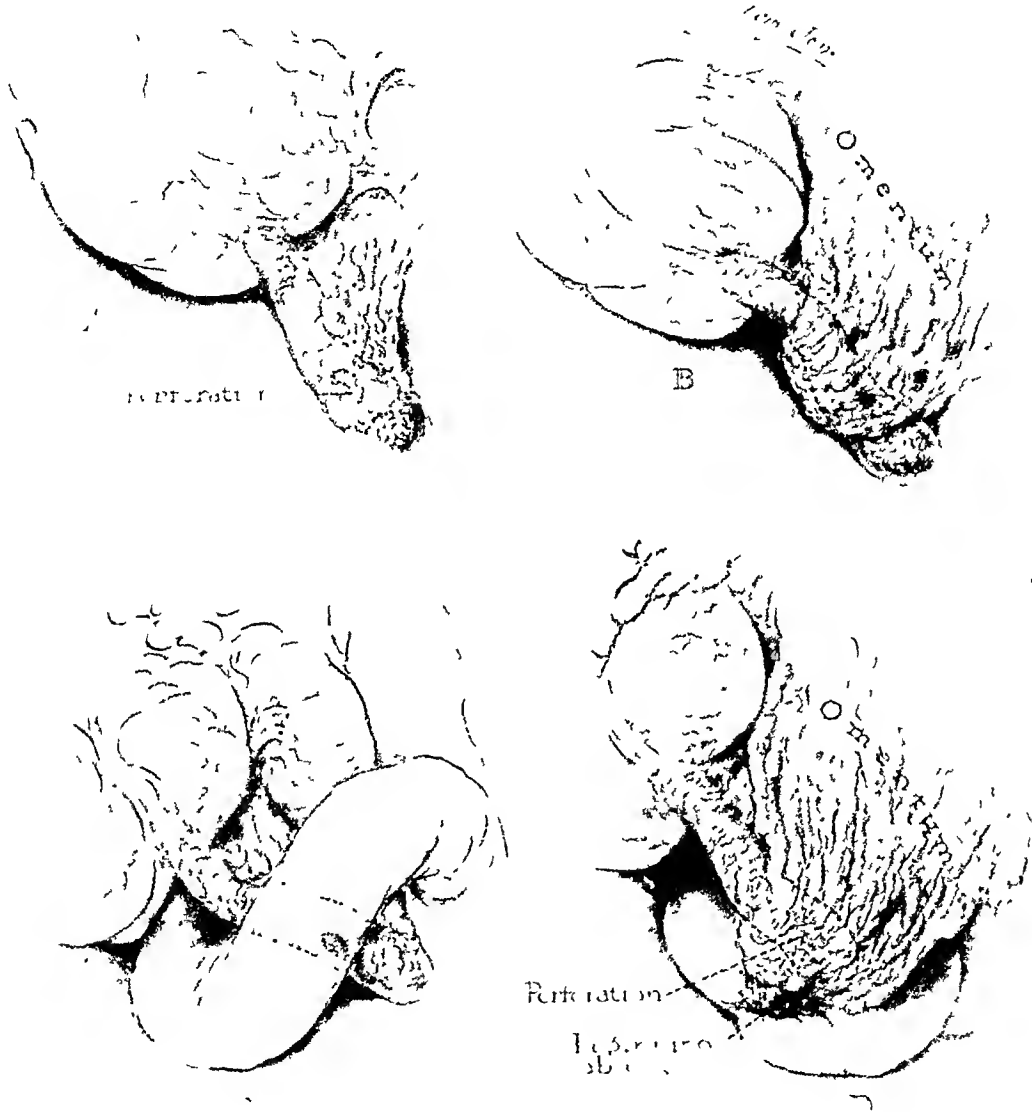
FIG. 1.

many instances, the assisting intern, whose vision of the gross intraperitoneal changes has been more limited than that of the operator, is directed to describe the gross findings on the chart. His description invariably is limited to the gross changes affecting the appendix. Neither he nor the operator suspects that a localizing process has just been converted into a frank spread-

operation because of "meddlesome surgery" he has had projected into his capillaries and lymph vessels a tremendous amount of antigen.

A diagnosis of peritonitis complicating acute appendicitis is made in less than 5 per cent of instances by the attending physician before the patient is hospitalized, despite the fact that over 30 per cent of these

patients have perforated appendices before admission. Surgeons generally have little difficulty in diagnosing a frank perforation, but the incipient perforations are frequently not so diagnosed preoperatively.

More accurate preoperative diagnoses will help reduce the high mortality of spreading peritonitis, which, when diagnosed, calls for scientific management rather than immediate operation.

2. *The Important Rôle of Immunity— Local Tissue and General—in the Management of Spreading Peritonitis.* Nature has but one method of combating infections regardless of location or origin. What happens to a patient following bacterial invasion of normal tissue depends on the dose of antigen and his reactive capacity.

In an intact appendix the amount of antigen absorbed is sufficient to induce normal antibody formation only to a minimal degree.[2] My associates and I demonstrated the presence of antitoxin for the Clostridium welchii toxin in the blood serum of patients who had recovered after operation for acute appendicitis in only 20 per cent of instances, while the serum of those operated upon for spreading peritonitis due to a perforated appendix showed antitoxin in 72 per cent of instances. When the serous coat of the appendix is intact, the amount of antigen necessary to induce humoral antibody formation is approximately .000004 mg.[3] If clasmatocytes have anything to do with the formation of antibodies, and certain investigators[4,5,6,7] think they do, then the development of a local tissue immunity is interferred with when the appendix is removed or even when drainage is instituted before a localizing or spreading process has been allowed to abscess.

The time which elapses between invasion of tissue by micro-organisms and operation must be considered if patients are to be managed properly. Agglutinins in large quantities do not appear in the blood of patients or animals following invasion by the typhoid bacillus before the seventh day.

Antitoxin to the C. tetani has been observed in local abscess in hyperimmunized animals on the seventh day. Patients resistant to the invasion of the lungs by the pneumococci show antitoxin in great quantities directly preceding or coincident to the crisis. My associates and I have not been able to demonstrate antitoxin to the Clostridium welchii in the blood serum of patients who have recovered from spreading peritonitis due to a perforated appendix before the sixth day.

3. *Correlation of Symptoms, Signs and Gross Changes at Operation with What Happens to the Patient.* Surgeons, like all inhabitants of the globe, have been caught in the "swirl of hurry." In an effort to expedite the performance of surgical procedures, much has been sacrificed—the policy of get in and get out quickly has resulted in a lack of careful observation. A review by the author of many thousands of clinical records of patients operated upon for acute appendicitis and its complications failed, with very few exceptions, to disclose descriptions of pathologic changes in the parietal peritoneum, not only in acute appendicitis but in local and spreading peritonitis as well. To a lesser degree this was also true of the gross changes in the visceral peritoneum beyond the central zone of infection. This failure to describe is due in part to an obsession on the part of some surgeons to be reputed fast operators; but also to incomplete visibility of the pathologic process. When operating for acute appendicitis and its complications, surgeons use the small transverse or McBurney incision in approximately 37 per cent of instances. Regardless of the type of incision, the appendix presents itself uncovered at the opening in the peritoneum in less than 10 per cent of instances. Usually, omentum or intestine must be displaced before it can be located and then frequently only a part of it may be visible. If it can be seen, the most important procedure up to this point can be carried out: the inspection of the serous coat for perforation. If it cannot be seen,

the next step, the visualization of the appendix is proceeded with.

How important is this next step and what technique is usually employed? The average surgeon does one of two things: he places gauze strips in the peritoneal cavity and gently pushes aside the coils of intestine or the omentum until he sees the appendix; or he inserts his index finger through the peritoneal opening in the direction of the pelvic brim and sweeping it upward, hooks the appendix in the crook of the finger. The latter procedure is legitimate if the appendix is not an over-distended gangrenous or gangrenosuppurative type or if the process is not a localizing one. The percentage of intact appendices that are ruptured in this manner is not large—the percentage of cases in which the localizing process is converted into a spreading one is larger than can be considered moderate by any surgeon. A correct correlation of gross changes found at operation with symptoms and signs preceding cannot be made by the surgeon because the perforated and at times, leaking appendix which he exposes is not the cause of these symptoms and signs. The condition in which he leaves the peritoneal cavity, however, is the cause of what happens after operation. Our surveys show a mortality of 20 per cent in cases where a perforated appendix is removed during the *localizing* stage but in twelve hospitals where appendices were removed routinely in the presence of spreading peritonitis, the mortality was 37.5 per cent.[8]

Surgeons and clinical pathologists must therefore, correlate symptoms, signs and gross changes observed at operation with functional variations in the living, thereby supplying the medical profession with data which will make possible a more accurate diagnosis.

SUMMARY

A reduction of the excessively high mortality of spreading peritonitis complicating acute perforative appendicitis will occur if and when symptoms, signs and gross changes found at operation are correlated with what happens to the patient. It is *not* what is found at postmortem, that is important in the recovery of patients. Local tissue and general immunity play an important rôle.

A clinical pathologic classification of acute appendicitis and peritonitis complicating perforative appendicitis embodying these essential features is presented.

REFERENCES

1. DEAVER, J. B. Appendicitis—Its History, Anatomy, Clinical Pathology, Diagnosis, Treatment, Technique of Operation, 4th Ed. Phila., 1913. P. Blakiston Son & Co.
2. BOWER, J. O., MENGLE, H. A., and PAXSON, N. F. Demonstration of antitoxin for clostridium welchii toxin in the blood serum of patients and dogs that have recovered from spreading peritonitis complicating acute perforative appendicitis. J. Immunology, 34: 185, 1938.
3. BANHAM and HUMPHREY. Amount of antigen and death. J. Bacteriol., 13: 46, 1927.
4. TOPLEY, W. W. C. An Outline of Immunity. Baltimore, 1933. Wm. Wood.
5. GAY, F. P., and MORRISON, L. F. J. Infec. Dis., 33: 338, 1923.
6. ROBERTSON, O. H., and WILEY, C. G. J. Clin. Investigation, 15: 1, 1937.
7. CANNON, C. R., and PACHERO, G. A. Ann. Surg. Path., 6: 749, 1930.
8. BOWER, J. O., BURNS, JOHN C., and MENGLE, H. A. Induced spreading peritonitis complicating acute perforative appendicitis. Surg., Gynec. & Obst., 66: 947, 1938.

FURTHER EXPERIENCE WITH MERCRESIN*

WALTER G. MADDOCK, M.D.   AND   LUCILLE K. GEORG, M.S.

ANN ARBOR, MICHIGAN

THE importance of the normal bacterial flora of the patient's skin as a possible means of wound infection has been emphasized by many workers.[1] As a result, the bactericidal efficiency of the various germicides used in preoperative skin disinfection has been extensively studied.[2,3,4,5,6,7,8,9] The laboratory evaluation tests of these germicides have been standardized by the United States Food and Drug Administration,[10] but the clinical evaluation has offered considerable difficulty. The conditions of individual resistance, immunity, differences in kinds and virulence of the organisms, the relative sterility of the air in the operating room,[11] the type and extent of the operation, and the surgical methods employed are variable factors which make definite conclusions as to the relative merits of one germicide compared to another very difficult. In some investigations the clinical evaluations of the various germicides have dealt with artificial conditions such as in animal skin disinfection tests, the application of various laboratory cultures of bacteria to the surface of the skin, the painting of a very small area of skin under conditions not similar to those encountered in the operating rooms, and studies which take into consideration only the superficial contaminants of the skin.

In order to determine the bactericidal efficiency of several germicides under conditions of use in the operating rooms of the University Hospital, a series of studies were carried out. The following questions seemed to be of practical importance:

1. Is the skin of the operative area contaminated when the patient is brought into the operating room?

2. Can the skin of the operative area be sterilized by a germicide preparation as employed in the operating room?

3. Does the germicide penetrate the skin and sterilize the deeper layers?

4. Does the skin remain sterile throughout the operation?

5. Is there a difference in the bactericidal efficiency of the various germicidal preparations studied?

One hundred and seventy-three operative cases were selected for study. These included herniorrhaphies, appendectomies, cholecystectomies, gastric resections, etc. Because most of these cases were complicated (the hernias often were bilateral or required fascial sutures; the patients with biliary tract disease needed exploration of the common duct), the average duration of the operation was one and a half hours. The germicides studied for skin preparation were: (1) tincture of mercresin 1:1000 (ortho-hydroxyphenylmercuric chloride and a synthetic cresol); (2) tincture of merthiolate 1:1000 (sodium ethylmercurithiosalicylate); (3) tincture of iodine 2 per cent U.S.P. followed by 50 per cent propanol.

METHOD

The routine preparation of the operative field before the patient was brought to the operating room was as follows: On the day preceding the operation the operative area was scrubbed with tincture of green soap and shaved. This was followed by sponging the area with 2 per cent boric acid solution, then with ether and finally with 50 per cent propanol. A sterile towel or dressing covered the area overnight. On the morning of the operation the operative field was again carefully washed with ether followed

---

by 50 per cent propanol and a sterile dressing again applied.

The technique used in the experimental work was as follows:

1. Control cultures were made from the skin of the operative area when the sterile dressings were removed in the operating room. The cultures were taken by rubbing two sterile swabs, moistened with sterile broth, over the skin surface. One of the swabs was transferred immediately to a tube containing 10 c.c. of sterile dextrose broth. The second swab was cultured anaerobically by breaking it off aseptically into a tube containing 10 c.c. of freshly boiled milk, and then excluding air from the medium by pouring hot sterile paraffin over the surface of the liquid.

2. Two swab cultures were made from the skin of the operative area after the area had been sprayed (or painted, using sterile swabs) with the germicide, and the drapes had been placed about the operative field. The time between the application of the germicide and the completion of the draping varied from two to three minutes. One of the two swabs from the skin of the operative field was immediately transferred aseptically to a 250 c.c. Erlenmeyer flask containing 150 c.c. of sterile dextrose broth. The other swab was broken off aseptically into a large tube containing 80 c.c. of freshly boiled milk, and the surface of the milk was sealed over with sterile paraffin in order to exclude the air.

3. In ninety-one cases a small piece of skin was excised from the edge of the wound when the initial incision was made. Each of these pieces of tissue was ground in a sterile glass mortar with 5 c.c. of sterile saline solution and a small amount of sterile sand. The ground tissue suspension was then transferred to Erlenmeyer flasks containing 150 c.c. of sterile dextrose broth.

4. In 138 cases, two swab cultures were taken at the end of the operation, as soon as the skin approximation had been completed. One swab was transferred to an Erlenmeyer flask containing 150 c.c. of sterile dextrose broth and the other was broken off aseptically into a tube containing 80 c.c. of freshly boiled milk. The milk medium was then sealed with melted paraffin, as described above, to exclude the air.

All cultures were incubated at 37°C. for seven days. If there was any trace of clouding in any of the flasks or tubes, dextrose agar plates were streaked and incubated for five days. As soon as a plate showed evidence of bacterial growth, smears were made from all the types of colonies present. The smears were all stained by the Gram method and examined microscopically. If flasks showed no growth after seven days' incubation, inhibition controls were made by transferring a 4 mm. loop of a twenty-four hour culture of Staphylococcus aureus, grown in 10 c.c. of dextrose broth, to each flask. The flasks were then again incubated at 37°C. If there was no clouding of the broth after three days' incubation, that case was discarded, since it was assumed that sufficient germicide had been carried over on the swab to render the medium inhibitory to the growth of Staphylococcus aureus.

It had been previously determined by experiment that at least 100 c.c. of broth must always be used in the culturing of the germicide covered swabs in order to insure sufficient dilution of the germicide to permit growth of any organisms which might be present on the swab. The method of planting the swab in a tube containing 50 c.c. of broth, and then making a subculture after twenty-four hours' incubation by transferring 1 to 2 c.c. of this medium to a fresh tube of 50 c.c. broth was not successful. Either sufficient germicide was carried over to the subculture, or bacteria had been killed in the first tube by long contact with the weakly germicidal solution. Fifty c.c. of milk proved to be a sufficient quantity to dilute the germicide carried over on the swabs and make the medium non-inhibitive to the growth of anaerobic bacteria.

DATA AND COMMENT

In eighty-three operative cases, tincture of mercresin was used; in seventy, tincture of merthiolate; and in twenty cases 2 per cent tincture of iodine U.S.P. followed by 50 per cent propanol. Each germicide was employed in all of the various kinds of operations, and no one surgeon limited his preoperative skin preparation to any one of the three germicides.

In Table I the data on the sterility of the operative field before and after the application of the germicide are presented. The control cultures show (1) the total failure of the routine ward preparation to clear the field of aerobic organisms, and (2) the presence of anaerobes in about 10 per cent of the cases. After the use of the germicide 61 to 88 per cent of sterile aerobic cultures was obtained, with mercresin showing the best results. The anaerobic culture data showed slight improvement over the control series, but no significant difference between the germicides.

In Table II the tissue culture data show a higher degree of sterility with mercresin than with the other germicides. For the postoperative aerobic cultures, mercresin again gave the best results. Most of the unsterile fields with any of the germicides were found at the end of the longer operations. The postoperative anaerobic cultures were essentially the same as at the beginning of the operation.

In Table III the kinds of organisms found in all of the positive cultures are listed. No

TABLE I

STERILITY OF THE OPERATIVE FIELD BEFORE AND AFTER THE APPLICATION OF THE GERMICIDE

Germicide Preparation	Swab Cultures from Skin Surface of Operative Field											
	Before Preparation with the Germicide (Control Cultures)						After Preparation with the Germicide					
	Aerobic			Anaerobic			Aerobic			Anaerobic		
	+	−	Per Cent Sterile	+	−	Per Cent Sterile	+	−	Per Cent Sterile	+	−	Per Cent Sterile
Tincture mercresin 1:1000	83	0	0	11	72	87	10	73	88	5	78	94
Tincture merthiolate 1:1000	70	0	0	8	62	88	27	43	61	2	68	97
Tincture of iodine 2 per cent U.S.P. & 50 per cent propanol	20	0	0	2	18	90	5	15	75	0	20	100

TABLE II

SUMMARY OF TISSUE CULTURE AND POSTOPERATIVE CULTURE DATA

Germicide Preparation	Tissue Culture. Culture of Macerated Piece of Skin Excised from Edge of Wound			Postoperative Cultures. Swab Cultures Taken from the Skin Surface at the End of the Operation					
	Aerobic			Aerobic			Anaerobic		
	+	−	Per Cent Sterile	+	−	Per Cent Sterile	+	−	Per Cent Sterile
Tincture of mercresin 1:1000	8	27	77	32	36	54	1	67	98
Tincture of merthiolate 1:1000	22	19	46	34	16	32	3	47	94
Tincture of iodine 2 per cent U.S.P. & 50 per cent propanol	5	10	66	12	6	33	0	18	100

TABLE III

ORGANISMS IDENTIFIED FROM POSITIVE CULTURES

	Per Cent
Staphylococcus albus	59
Gram-positive bacilli	19
Gas-producing anaerobes	6
Staphylococcus aureus	4
Diphtheroids	4
Sarcina	3
Gram-negative bacilli	2
Diplococci	1.5
Streptococci	1

significant variation occurred in the kinds of organisms isolated when any one of the three germicides was used.

CONCLUSIONS

From a study of operative field sterility under routine conditions used at the University Hospital, the following facts were found:

1. Routine pre-operating room preparation did not render any operative field free from aerobic bacteria. In about 10 per cent of the cases anaerobes were also present.

2. Germicides applied in the operating room sterilized the surface of the operative field in a high percentage of cases.

3. Full-thickness pieces of skin cut from the edges of the wound were sterile in about 10 per cent fewer cases than found for the surface study.

4. As the time period of the operation lengthened more positive surface cultures were obtained.

5. Tincture of mercresin gave the best results when compared to two other germicides.

REFERENCES

1. Sutton, H. B. Inadequate skin preparation as cause of postoperative wound infection. *New York State J. Med.*, 28: 129, 1928.
2. Birkhaug, K. E. Phenyl-mercuric-nitratc. *J. Infect. Dis.*, 53: 250, 1933.
3. Marks, H. H., Powell, H. M., and Jamieson, W. A. Merthiolate as skin disinfecting agent. *J. Lab. & Clin. Med.*, 17: 443, 1932.
4. von Oettingen, W. F. Comparative studies on mercurochrome and other antiseptics. *J. A. M. A.*, 99: 127, 1932.
5. Roberts, K. Value of methylene blue-gentian violet 5 per cent, in preoperative skin preparation. *Ann. Surg.*, 89: 183, 1929.
6. Scott, W. W., and Birkhaug, K. E. Comparative value of metaphen in alcohol-acetone-aqueous solution in preoperative disinfection of skin. *Ann. Surg.*, 93: 587, 1931.
7. Yanovskaya, A. P. Bacteriologic examination of skin in operated patients. *Vestnik kbir.*, 37: 159, 1935.
8. Ehlert, H. Vergleichende Studien zur Hautdesinfektion. *Zentralbl. f. Chir.*, 62: 2770, 1935.
9. Maddock, W. G., Boyden, A. M., and Malcolm, R. L. Mercresin (amyl cresolmercury preparation). A new surgical germicide. *Am. J. Surg.*, 34: 47, 1936.
10. Ruehle, G. L. A., and Brewer, C. M. United States Food and Drug Administration methods of testing antiseptics and disinfectants, U. S. Dept. of Agriculture, Washington, D. C., Circular #198, 1931.
11. Hart, D. Sterilization of air in operating room by special bactericidal radiant energy. *J. Thorac. Surg.*, 6: 45, 1936.

A COMPARISON OF THE EFFECTS OF A STERILE ANTISEPTIC JELL WITH AND WITHOUT LARVAL INGREDIENT ON WOUNDS

AN EXPERIMENTAL INVESTIGATION

GEORGE MILLES, M.D. AND ROGER T. FARLEY, M.D.

CHICAGO, ILLINOIS

MAGGOT therapy for all its merits has found limited application because of lack of ready availability of live, sterile maggots, and because of patients' psychological reaction to it. A number of workers have attempted to circumvent these objections by using two general methods of approach. The one has attempted to determine the beneficial effective principle.[1] The other has applied the whole macerated larvae or saline extracts prepared from the whole macerated larvae.[2,3,4]

LARVAL JELL

In order to overcome the difficulties encountered clinically by prior workers, a jell was prepared which incorporates comminuted whole larvae in a bland, water-soluble medium to which are added antiseptic, analgesic and preservative factors. This jell contains 5 per cent comminuted larvae Lucilia sericata, chlorbutanol and oxyquinoline sulphate,[5] a nontoxic antiseptic mixing compatibly with proteins.

This preparation is not burdened by the deterrent influence of oil bases, and permits the larval substance to come in close contact with the tissues. The vehicle prevents deterioration. Acidity is controlled to a low pH at which bacterial growth is inhibited, and the product contains inhibitory antiseptics which combine with the comminuted proteins present without precipitating them.

EXPERIMENTS

In order to compare the influence of the jell above described with a similar one without comminuted larvae, the following experiments were undertaken. Symmetrical and as nearly as possible identical wounds of various types were inflicted on the shaved upper backs of twenty-six rabbits, under suitable anesthesia. To the wound on one side the plain jell containing antiseptic, analgesic and preservative elements was applied, and to the wound on the opposite side the jell containing the comminuted larvae was applied in similar manner. Daily observations were made on the gross appearance of the wounds. At various stages of healing the animals were sacrificed and the traumatized tissue was sectioned for comparative microscopic examination.

Burns. The skin was burned with a red-hot iron on both sides to approximately equal extent, producing third degree burns on eleven animals.

The respective medications were administered continuously during the entire period of observation.

Gross Appearance. (Rabbit No. 20.) During the first four days both wounds had the same necrotic appearance. An elevation in the center of the larval treated wound decreased gradually, and a serous exudate appeared which differentiated it from that treated with the plain jell. On the ninth day the larval treated side showed evidence of beginning repair. On the eleventh day granulation appeared, which showed a tendency to ooze. Meanwhile the wound treated with the plain jell hardly changed until the thirteenth day, when granulations first became evident. These differences persisted until the sixteenth day when the animal was sacrificed. The wound treated with the larval jell at this time was covered with healthy granulation, and showed beginning epithelization. The wound treated with the

plain jell showed a less pronounced tendency to heal.

Microscopic Examination. Section of the

débris, but in which there was surprisingly little evidence of granulation tissue formation or cellular proliferation in general and in which

FIG. 1. Rabbit 4. Slide 7. Maggot.

(non-larval side) through the margin of an ulcer which extended almost through the subcutaneous muscle showed a sharp line of demarcation between the outer necrotic layer, which was diffusely infiltrated with leucocytes

the healing response was very small. The epithelial margin of the ulcer displayed some evidence of proliferation.

Another slide showed the depth of the ulcer (larval treated) approximately the same as in the

FIG. 2. Rabbit 4. Slide 8. Non-maggot.

and retained only the shadows of a preëxisting structure, and a deep layer of normal tissue. The two graded into an intermediate zone diffusely infiltrated with leucocytes and cellular

preceding. The surface was covered with a crust of leucocytes and granular débris. The subcutaneous tissue displayed considerable evidence of connective tissue proliferation in the

form of fibroblasts and a rather marked leucocytic infiltration. This response was noted especially along the line between the dead and living

FIG. 3. Rabbit 10. Slide 11. Non-maggot.

tissue. The marginal epithelium displayed only a very limited proliferative response, though along the margin the cells were hyperplastic and occasional mitotic figures could be seen. In comparison with the former slide, there appeared to be a better healing response throughout the ulcer.

Gross Appearance. (Rabbit No. 4.) By the sixth day the wound treated with the larval jell, in addition to being lighter and moister, showed a fading of the line of demarcation aground the wound and merging with the surrounding normal skin. On the seventh day the wound treated with the larval jell exhibited a return of circulation in the burned area and, on the tenth day, when the animal was sacrificed, gave the impression of reparative processes extending from the surrounding tissue into the burned area. The wound treated with the plain jell still had a definite line of demarcation, raised edge, and yellowish-gray sluggish appearance.

Microscopic Examination. On the non-larval side, section through an ulcer displayed a rather narrow epithelial margin in which proliferation was active. The floor of the ulcer was made up of collagen-rich connective tissue with some scattered islets of blood vessels and fibroblasts. The picture was that of a chronic ulcer.

Another section was quite similar, with, however, much more extensive evidence of connective tissue proliferation in the floor of the ulcer. The zone of epithelial hyperplasia was also wider. In comparison with the preceding section this slide showed active healing rather than the indolent healing which characterized the preceding ulcer.

Non-Infected Wounds. The full thickness of skin was aseptically removed, exposing the underlying muscle in four animals. The dressings were applied immediately. A typical protocol is given:

Gross Appearance. (Rabbit No. 21.) During the first four days the wound treated with the plain jell was bloody, with the edges free except for the medial segment.

The wound treated with larval jell was completely covered with a grayish-white membrane, and the edges were closely adherent to the deep fascia. On the sixth day the edges of the wound treated with larval jell were granular. The next day the wound was dark red and the granulations were less pronounced. This difference continued until the eleventh day when the animal was sacrificed. The wound treated with the plain jell was only one-fourth filled with granulations, while the wound treated with the larval jell was completely filled with active granulation tissue oozing on pressure.

Microscopic examination of the non-larval side showed the epithelium hyperplastic along the margin of the ulcer and for some distance

Fig. 4. Rabbit 10. Slide 12. Maggot.

back. The surface of the ulcer was covered with a crust of granular débris diffusely infiltrated with leucocytes. Beneath this was a layer of granulation tissue consisting of fibroblasts, young connective tissue cells and numerous blood vessels. This zone was the seat of a scattered round cell infiltration and ended rather sharply in the normal subcutis.

Another section was made through an ulcer, the epithelial margin of which displayed healthy proliferation for some distance back. The ulcer was incompletely covered by a necrotic crust, beneath which lay a zone of granulation in which collagen tissue formation was fairly well marked. The proliferation in this zone was prominent in comparison with the preceding section; healing appeared to be somewhat more advanced, i.e., this ulcer was less indolent than that in the preceding slide.

Infected Wounds. The skin was lacerated and inoculated with a virulent culture of Staphylococcus pyogenes aureus in ten animals. Since rabbits are very resistant to such infections, in many cases more than one inoculation was made to obtain a suitably infected wound. In all cases treatment was begun only after the desired degree of infection was approximately equal in both wounds.

Gross Appearance. (Rabbit No. 10.) The first day after application of the jell, the wound treated with larval jell developed a line of tissue repair visible to the unaided eye. There was considerably more induration at the edges of the wound treated with plain jell. The third day, after necrotic débris was removed, the wound treated with larval jell appeared filled with granulation. The wound treated with plain jell was covered with a gray-yellow necrotic membrane. The observations are terminated on the fifth day when the wound treated with larval jell was almost healed, while the wound treated with plain jell was still necrotic.

Microscopic examination of a non-larval section through an ulcer showed an epithelial margin of active proliferation. The floor of the ulcer was made up of collagen-rich scar tissue with moderate evidence of connective tissue proliferation in the form of scattered islets of fibroblasts. Leucocytic infiltration was rather limited. The section displayed a well advanced stage in the healing process.

A second section resembled the first closely, but the evidences of proliferation in the connective tissue were somewhat more marked and extended rather deeper. The epithelial margin displayed a more active picture of proliferation. This section also displayed an advanced stage of healing and the ulcer was less indolent.

Gross Appearance. (Rabbit No. 14.) Three days after the original application of the mate-

rials, the wound treated with larval jell showed evidence of closing, and on the fourth day healthy granulations sprang from the edges. On

tion tissue. By the twelfth day the area treated with larval jell was completely covered with granulation tissue and dry, while the wound

FIG. 5. Rabbit 20. Slide 16. Non-maggot.

the sixth day this wound was nearly filled with granulation tissue. The wound treated with plain jell was beginning to fill, but still showed considerable necrosis. On the ninth day the

treated with plain jell was still moist and contained a slight amount of necrotic tissue. On the sixteenth day when the experiment was terminated, the wound treated with plain jell

FIG. 6. Rabbit 20. Slide 15. Maggot.

wound treated with the larval jell was not indurated and had completely closed, showing a slight amount of granulation tissue in the central area. The wound treated with plain jell was still draining and contained less granula-

was only three-fourths filled with granulations and still draining.

Microscopic examination (non-larval side) was not entirely satisfactory for our purpose since it did not adequately depict the floor of the

ulcer. However, the covering epithelium was present as a rather narrow layer which merged suddenly into a small zone bordering the ulcer.

displayed marked evidence of proliferation up to the ulcer. The surface was covered in part with a narrow crust. The floor was made up of

FIG. 7. Rabbit 21. Slide 14. Non-maggot.

In this area the epithelium was rather atrophic, its cells being small, with small, deeply stained nuclei, almost devoid of evidences of hyperplasia. The base of the ulcer was narrow and

young connective tissue, in which some collagen fibrils could be recognized, diffusely infiltrated with leucocytes. The stroma was vascular and healing was well advanced. In comparison with

FIG. 8. Rabbit 21. Slide 13. Maggot.

made up of collagen-rich connective tissue cells whose nuclei were small, spindle-shaped and pyknotic. There were but few round cells present.

A section through a narrow ulcer included both epithelial margins. The epithelial margins

the first slide the evidences of healing were much more marked, especially in the marginal epithelium.

Chemical Burns. In the fourth series, only two animals were used since the

wounds were much more violent than anticipated. One-half Gm. of sodium hydroxide was placed on each shaved side and permitted to remain in situ for twenty-four hours. The thick skin was completely destroyed over an area about 1½ inches in diameter; the surrounding skin was dark and necrotic, and the underlying muscle dark, necrotic, and partially destroyed in two cases, exposing the scapulae. The wounds were so extensive that the animals lost about a fourth of their weight and were not expected to live, but continuous treatment resulted in healing of the wounds and restoration of normal health.

Gross Appearance. (Rabbit No. 22.) By the fourth day the wound treated with the larval jell was brighter red than the one treated with the plain jell, which still appeared completely necrotic. The first evidence of granulation showed in the skin surrounding the deep crater, more noticeably in the side treated with larval jell than in the other. Both wounds discharged a serous exudate. By the ninth day the wound treated with larval jell showed evidence of muscle repair. By the fifteenth day much necrotic material had been removed from both wounds, but the one with plain jell remained necrotic and was healing with a slight discharge. The discharge from the larval treated wound was more abundant. The floor had a healthy granular appearance, and the surrounding induration was not so great. On the eighteenth day the larval treated wound was filling rapidly, and was surrounded by an actively proliferating ring of new epithelium. Cn the twenty-fifth day the larval treated wound was practically filled with granulation tissue, while the other still appeared necrotic. The experiment was terminated on the thirty-fifth day, when the larval treated side was dry and completely filled with pink granulation tissue while the one with the plain jell had not as yet completely lost its necrotic appearance.

Microscopic Examination. The section was made up of a connective tissue stroma from the non-larval side, scattered through which were several variably sized sharply circumscribed zones occupied by pink stained granular necrotic débris and pus cells.

The stroma was quite vascular and varied somewhat in relation to the abscesses described.

Immediately surrounding them the stroma was rich in fibroblasts and in some instances epithelial cells were seen. As the distance from these areas increased, the stroma cells became more mature, though generally hyperplastic in type, and the collagen fibrils increased progressively. The stroma was the seat of a variable scattered infiltration of neutrophiles, eosinophiles and round cells.

The picture was that of an active "healthy" response to a well localized infection with abscess formation.

Another section through the ulcer did not include the abscess spaces in the previously mentioned one. The floor of the ulcer was made up of fairly old proliferating connective tissue, *moderately vascular and containing a moderate* amount of collagen. Cellular infiltration was rather limited. An outer covering of necrotic material was present in part of the section. This section showed a good "healthy" healing response.

Gross Appearance. (Rabbit No. 23.) By the fourth day the larval treated wound showed some evidence of tissue regeneration while the wound treated with plain jell was still much more necrotic. By the seventh day the surrounding skin on the larval treated wound showed beginning epithelial proliferation, but the other wound had just begun to granulate. On the ninth day underlying muscle in the larval treated side showed evidence of repair. By the twelfth day on the larval treated side most of the necrotic débris was removed, exposing granulation throughout, while the wound treated with plain jell was still covered with a necrotic membrane. In both wounds granulation tissue grew exuberantly, almost to the point of tumefaction. On the larval treated side this excessive granulation lessened within a few days and the wound began filling; in the other wound the excessive granulations persisted. On the twenty-fifth day the experiment was terminated. The larval treated wound was practically filled with active granulations. The wound treated with plain jell still contained necrotic tissue, and was surrounded by a sluggish, indurated granulating edge.

Microscopic examination of the non-larval side was done. The section was through the margin of a cutaneous ulcer including the floor. The covering epithelium faded off into the ulcer, grading from a zone of epithelial hyperplasia, indicated by the large vesicular nuclei

and occasional mitotic figures, through one of increasing intercellular edema to a strand of atrophic epithelial cells which finally ended in

The epithelial margin of the ulcer in a second slide displayed a somewhat more marked proliferative reaction. The narrow layer of
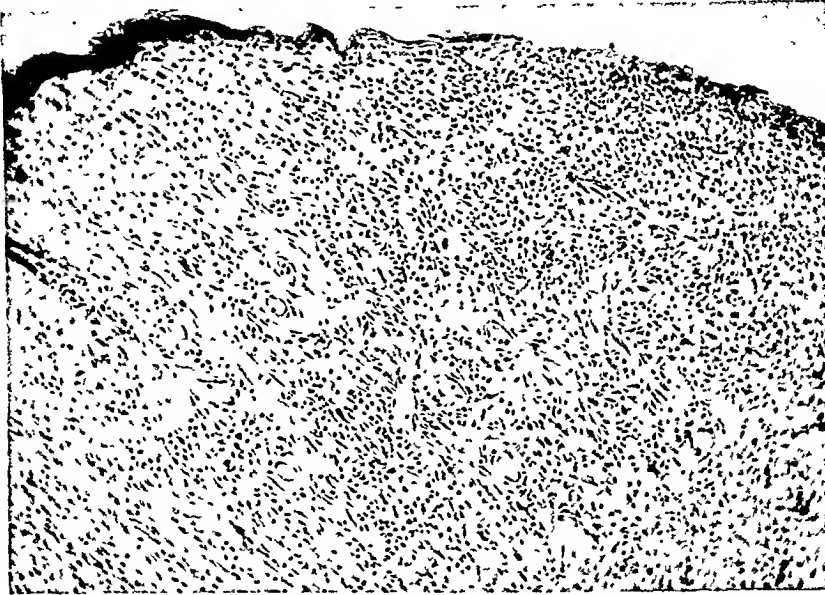
Fig. 9. Rabbit 23. Slide 29. Non-maggot.

the ulcer. The ulcer floor was in part covered by a necrotic membrane. A narrow outer zone was made up of dense hyalinized fibers and beneath this lay a vascular edematous stroma of young

squamous epithelium immediately about the border of the ulcer was still present, however. The floor of the ulcer was made up of a rather compact fibroblastic stroma, displaying a mod-

Fig. 10. Rabbit 23. Slide 30. Maggot.

connective tissue cells, with a moderate diffuse infiltration of polymorphs, chiefly eosinophiles.

The picture was that of a moderately active proliferative response. There was relatively little leucocytic infiltration in the stroma.

erate collagen formation, except that the very surface was vascular and diffusely infiltrated with leucocytes. In comparison with the preceding slide, this section displayed a more advanced stage of healing, as indicated by the

more mature connective tissue making up the ulcer floor.

SUMMARY

1. A group of animals were bilaterally traumatized in order to observe the difference in the progress of healing under treatment with (a) a sterile antiseptic jell containing comminuted larvae Lucilia sericata; and (b) a similar jell without the larval material.

2. Observations were made on eleven rabbits with thermal third degree burns, four rabbits with non-infected wounds, nine rabbits with wounds infected with staphylococcus pyogenes aureus, and two rabbits with sodium hydroxide burns, a total of twenty-six animals.

3. Observations on the gross appearance of the wounds indicate more rapid healing in the wounds treated with the larval jell than in the wounds treated with the plain jell; in the more severe wounds this difference in healing was especially apparent.

4. Study of the microscopic sections, with a few exceptions, confirms the gross findings. In twenty-one rabbits the larval jell treated wounds displayed more active connective tissue proliferation and less evidence of infection than those with the plain jell.

5. The water-soluble jell (with and without comminuted larvae) used in the experiment was tested in accordance with the regulations of circular 198, December 1931, United States Department of Agriculture, and meets all the requirements of an antiseptic preparation set forth in that circular.

REFERENCES

1. LIVINGSTON and PRINCE. Treatment of chronic osteomyelitis with special reference to the use of maggot active principle. *J. A. M. A.*, 98: 1143, 1932.
2. LIVINGSTON. Therapeutic active principle of maggots with a description of its clinical application in 567 cases. *J. Bone & Joint Surg.*, 18: 751, 1936.
3. CUZIN. Use of extracts of larvae of Lucilia sericata in otorhinolaryngology and especially in treatment of ozena. *Soc. méd. mil. franç., Bull. mens.*, 29: 126, 1935.
4. PORTALES. Treatment of ozena by means of extract from "Lucilia sericata" larvae. *Med. ibera*, 1: 350, 1936.
5. United States Dispensatory, 22nd Centennial Edition, 1936, p. 1505.

GASTROINTESTINAL ULCERATION FOLLOWING BURNS*

JOHN L. KEELEY, M.D.

Harvey Cushing Fellow, Peter Bent Brigham Hospital; Research Fellow in Surgery, Harvard Medical School

BOSTON, MASSACHUSETTS

HISTORICAL

ALTHOUGH the ulcerations of the gastrointestinal tract which occur as a complication of cutaneous burns are commonly called Curling's ulcers, two other names are associated with the historical background of these lesions. Dupuytren,[1] in 1832, ten years before Curling's[2] article appeared, called attention to the changes in the "intestinal canal" which followed burns. Violent congestion, severe gastroenteritis, and "more or less vivid red patches and more or less deep ulceration" were the conditions he found, depending on the length of time the patient had survived the accident. Long,[3] in 1840, described two instances of fatal burns in which perforation of duodenal ulcers occurred.

Moynihan[4] gives priority to Long, but Novak[5] points out that "clearly recorded findings of gastrointestinal inflammation and ulceration after burns" were observed by Dupuytren in 1832. It appears that Dupuytren first described gastrointestinal ulceration after burns and as both Long's and Curling's patients had only duodenal ulceration, they may have felt that their cases were distinctly different from those of Dupuytren's. Novak, however, states, "It is difficult to understand this assumption if the latter ulcers were found under similar circumstances." Novak further calls attention to the case he reports in which the ulceration was confined to the stomach. He refers also to Leonard's[6] case in which ulcers were found in the stomach at autopsy four days after cauterization of the cervix by the Percy method. The case herein reported showed roentgenographic evidence diagnostic of duodenal ulcer while the fluoroscopic examination indicated ulcerative processes on both sides of the pylorus.

It is obvious that Curling did not believe he was reporting this lesion for the first time as he included as the ninth and tenth of his series two cases previously reported by Long, to whom he gave full credit. Furthermore, Curling states, "The circumstance of congestion in the mucous membrane of the alimentary canal in common with a similar condition of the blood vessels of the brain and lungs, occurring in the early stage of burns and of the stomach and intestines being subject to inflammation after recovery from the immediate effects of the injury were first particularly noticed by Dupuytren (reference given) but it does not appear that any suspicion was excited that the duodenum was the part most liable to suffer."

The facts that Curling's paper described lesions occurring specifically in the duodenum, as the title of his report indicates, and that his series is the largest one yet published have doubtless been responsible for the term "Curling's ulcer." That the lesions in the vast majority of reported cases have been similarly located has further established this term.

In the interest of accuracy it is suggested that the term "gastrointestinal ulceration following burns" be used except in those instances where the lesion answers the description originally given by Curling.

INCIDENCE

There have been comparatively few reported cases in approximately a century. Perhaps the best appreciation of the infre-

quency of this lesion may be gained from the following statistics. Perry and Shaw[7] report five cases found during the autopsies of 149 burn cases covering a period of fifty years, 1843–1892, at Guy's Hospital. Ronchese[8] cites one instance in 348 fatal burn cases. Bancroft and Rogers[9] report a series of 104 cases of cutaneous burns. Thirty-four per cent were under ten years of age. Twenty-eight per cent of the entire series were fatal yet no instance of gastrointestinal ulceration was noted. Levin[10] found this complication once in thirteen years in an autopsy service, doing 900 to 1,000 examinations annually. In addition he reported for two colleagues who had not seen a single case in ten and fifteen year periods respectively in similar necropsy services. Fenwick[11] reports that 6.2 per cent of all deaths from burns are due to complicating gastrointestinal ulceration. Harris,[12] in a series of 138 fatalities in 567 burn cases found one death due to this lesion. No instance of gastrointestinal ulceration is recorded among the 171 cases of burns or scalds seen in the Peter Bent Brigham Hospital during the period from 1913 to 1937. Seventeen of these cases terminated fatally.

ETIOLOGY

Curling suggested that Brunner's glands were called upon to perform the excretory functions for which the burned skin was incapacitated. He thought the resulting hypersecretion caused congestion and ulceration in the duodenum. Hunter[14] advanced the theory that in burns a toxic substance, secreted with the bile, was responsible for gastrointestinal ulceration. By the injection of toluylendiamin he produced duodenal ulcerations in dogs and believed this represented an analogous process. Moynihan[4] states that Fenwick secured similar results by the same methods when the dog's common bile duct was ligated. The article by Fenwick[15] to which Moynihan refers does not contain that statement, however. The embolic

theory has been said to be supported by Moynihan. His belief in the etiology of gastrointestinal ulceration following burns is expressed as follows: "Duodenal ulcer in connection with burns is doubtless a toxic ulcer . . . A point which requires investigation in this connection concerns the presence and possible influence of septic emboli . . . In the alimentary canal they would produce hemorrhagic infiltration which immediately beyond the pylorus would readily be converted into ulcers by the action of the gastric juice." He expresses the opinion that gastrointestinal ulceration does not occur unless sepsis is present in the burned areas.

Kapsinow[16] states, "As a result of extensive blood concentration, stasis with rupture of the capillaries underlying the duodenal mucosa producing an inefficient oxygen supply to the tissue, causes a necrosis of the mucosa and the typical picture of Curling's ulcer."

The theory which has received considerable attention recently is based on the association of cutaneous burns and adrenal damage. This was pointed out by Weiskotten[17] who demonstrated that swelling of, and hemorrhage into, the suprarenal medulla were rather constant findings in fatal cases of severe cutaneous burns. It has long been known (Mann,[18] Elliott,[19] and others[20,21]) that adrenalectomized dogs develop peptic ulcers which are acute in character. McLaughlin[22] by partially damaging both suprarenal glands with the electrocoagulating current, has produced intestinal ulceration in seventeen of a series of twenty-two dogs.

As in ordinary gastric and duodenal ulceration, the question of a neurogenic etiologic factor arises. For this reason McLaughlin performed another series of experiments.[23] He sectioned the vagi at the esophageal hiatus and divided the right vagus in the neck as a further guard against vagal influence to the upper gastrointestinal tract. Electrocoagulation of the adrenal glands produced ulceration similar to that seen in the' first series of experi-

ments with success in approximately the same proportion of instances.

Rokitansky[24] was probably the first to

Paek[36] states that occasionally an effusion of blood is found between the dura mater and the skull. Recently Globus and

FIG. 1. Film taken August 14, 1937 showing deformity of duodenal cap.

FIG. 2. Another film of the same date showing barium remaining in ulcer crater.

propose the neurogenic theory of gastrointestinal ulceration. The famous Virchow, by expressing his belief in a local cause of ulcer, diverted much scientific attention from Rokitansky's view. Cushing[25] revived considerable interest in the association of gastrointestinal ulceration and brain lesions by reporting eleven cases. Subsequent articles[26,27] have furnished additional instances of this combination.

It is true that hypophysectomy and experimental lesions in other parts of the brain are followed by gastric hemorrhages, erosions, ulcers and perforations.[28,29,30,31,32] The influence of vagotomy and of excision of the celiac plexus, the celiac ganglion and the abdominal portion of the sympathetic chain in various combinations prior to brain injury have been studied.[31,33] If brain damage in these areas is the cause of gastrointestinal ulceration after burns, one should expect to find some evidence of it. The fact remains that those who have studied the pathology of burns have either found no demonstrable changes in the brain[34,35] or have detected, with very few exceptions, nothing more than simple hyperemia.[36]

Bender[37] have reported an instance of disseminated degenerative encephalopathy secondary to extensive and severe burns. In general, however, demonstrable changes in the brains of burned patients represent the exceptions rather than the rule.

In a recent experimental study of burns[38] in profoundly anesthetized animals, intestinal erosions and ulcerations were present in two of the series of seven, yet the brains showed only those changes which could be justly attributed to the effects of prolonged anesthesia. It must be remembered, however, that in relatively acute processes impairment or alteration of function may occur before structural changes · can be demonstrated.

Interesting as it is to speculate in regard to the rôle of neurogenic influences in the etiology of gastrointestinal ulceration after burns, the evidence seems to favor the hemoconcentration theory of Kapsinow if a single etiologic factor is to be selected. In burned patients and in polycythemia vera the blood shows a rise in hematocrit with a normal protein content per unit volume of plasma. The higher incidence of peptic

ulcer in patients with polycythemia vera[39] than in general clinic population and the known tendency of polycythemic blood to form thrombi suggests that hemoconcentration is a very significant factor. It occurs early in burn cases due to loss of plasma into and from the burned areas. Its occurrence later in the illness may be due to the adrenal damage as hemoconcentration has been shown to be a manifestation of adrenal insufficiency.[40]

PATHOLOGY

The lesions, which have also been reported in cases of scalds,[10,12] are similar to acute ulceration of the gastrointestinal tract from other causes. They may be single or multiple, the former predominate in the duodenum while the latter are more frequently found in the stomach. Almost always the site affected is the duodenum between the pylorus and the ampulla of Vater. In the stomach the prepyloric region is involved in a manner similar except in number to that of ordinary gastric ulcer.

The lesions vary in size from 1 to 25 mm. in diameter. Hemorrhage, perforation, or healing indicates the course of the lesion. Novak[5] says that ulcers of this type do not become chronic, but the history in the case reported by Kirchmayr[43] is interesting in this respect. The patient, a boy, sustained a burn in 1907. Immediately upon his discharge from the hospital where the burn had been treated he began to experience epigastric pain which continued in paroxysms for twelve years. The complaint then became so severe that surgery was advised. At operation a large radiating scar was found in the horizontal position of the duodenum. Fluoroscopic and roentgenographic evidence indicating gastric and duodenal ulcers has been demonstrated in the case reported here, three years after the burn and the occurrence of hematemesis.

SYMPTOMATOLOGY AND DIAGNOSIS

The time of onset of this complication varies from eighteen hours in Parfick's

case,[44] to the instance noted by Simpson[45] in which acute abdominal symptoms with fever, thought to be due to intestinal ulceration, occurred 100 days after the burn. Ulcers were demonstrated at autopsy in Curling's patients, six and twenty-one days after the accident. Dupuytren's case was found to have intestinal ulceration at autopsy thirty-six hours after sustaining the burn. The case described in this report had hematemesis on the fourth day. It had been preceded by nausea on the first day and abdominal distress on the second day. In the more recent literature (Fitzgibbon,[46] Miller and Rubenstein,[47] Clements,[48] Levin,[10] Laird and Wilkerson[49]) the average time of onset in adults has been about fourteen days. One of Fitzgibbons' cases began to have ulcer symptoms "months" after his burn. Roentgenograms showed a deformed duodenal cap. There had been no previous history of any gastrointestinal disturbance.

According to Maes,[13] gastro-intestinal ulceration as a complication of cutaneous burns is seen most frequently in children, but the majority of cases reported in the last five years have been in adults. Epigastric pain and tenderness are often present although the first evidence of ulceration may be hematemesis or melena. The occurrence of the former in a burned patient should arouse suspicion, while the appearance of blood in stools or vomitus is good evidence that gastrointestinal ulceration is present.

To conclude that hematemesis or melena in a burned patient is due to ulceration is justified in a large majority of instances. This has been shown in autopsies of fatal cases and by x-ray evidence in some of the recently reported instances of recovery. However, Swain[50] and D'Ewart[51] each report a fatality from burns and although there had been obvious bleeding from the gastrointestinal tract, no ulcer was seen at autopsy. "Congestion" and "engorged vessels" were reported. Because of the acute course followed by the great majority of these lesions, either healing or perfora-

tion soon occurs, and hence the chronicity which characterizes the ordinary peptic ulcer is seldom seen. For the same reason it may be difficult to demonstrate the lesion roentgenographically during convalescence.

TREATMENT

There seems to be no specific prophylactic régime of treatment for gastrointestinal ulceration following burns. The adequate treatment of the patient with burns as is currently practiced covers most of the factors which are considered in the etiology of this complication. Pain, a very important factor in the shock of burns, is treated early and promptly by opiates or other analgesics. Blood concentration, due to loss of circulating plasma, is ordinarily controlled by administration of adequate amounts of physiologic salt solution or a dextrose solution given intravenously or subcutaneously. It may be impossible to correct the hemoconcentration in this way. In some instances it may be necessary to restore serum proteins, or to alter the osmotic pressure of the blood, by the use of acacia.[52] The local treatment with eschar-producing solutions (such as tannic acid or gentian violet) prevents loss of plasma-like fluid from the burned area. In addition to the eschar which it forms, gentian violet has definite bactericidal power[53] which further decreases the possibility of infection.

When ulceration is suspected in a burned patient, ordinary medical ulcer treatment should be instituted. If the ulcer offers evidence of bleeding, the well-established routine of absolute rest for the patient and his stomach should be enforced. Blood transfusions, which may have been used earlier to replace serum protein loss, should not be used in hemorrhage except to prepare the patient for surgery. Two events may indicate operative interference during the acute stage. One is perforation, and the other is hemorrhage. In the former instance the process is so rapid that in most cases walling-off by adjacent viscera or omentum provides an ineffective barrier. In the latter instance the decision to operate requires the exercise of considerable judgment. Hemorrhage usually subsides spontaneously, but it must be remembered that the location of these ulcers in the duodenum predisposes to erosion of the pancreatico-duodenal artery which in the first three cases of Curling's series resulted in hemorrhage followed by death in fifteen to twenty-four hours. In either perforation or hemorrhage the degree of altered physiology due to the burn must be added to the risk of a grave surgical emergency.

CASE REPORT*

J. W., a white male, aged 26 years, was admitted to Mercy Hospital, Chicago, at 4 A.M., July 12, 1934. He was burned about the head, neck, trunk, and upper extremities. When seen in the emergency room the patient was in moderately severe shock. He was transferred to his room and there external heat was applied by means of lights. Morphine sulfate, ¼ gr. (0.016 Gm.), and physiologic solution of sodium chloride were administered subcutaneously. Further examination revealed first degree burns over the abdomen and chest and about the knees and lower thigh surfaces. Second degree burns were present on the back and neck, from the waistline to the occipital region and extending anteriorly to involve the sides of the thorax and the flanks. The skin of the face was involved to the same degree. The skin of the hands and lower forearms had been burned more deeply, third degree burns being found over these areas. The remainder of the physical examination was essentially within normal limits except for an old symblepharon on the left eye which was almost total, extending up to and including the pupillary area of the cornea. Vision in the left eye was reduced to light perception. The past history revealed no gastrointestinal complaints of any kind. The burned areas were sprayed with a 3 per cent solution of tannic acid and this procedure was repeated during the first few days until all areas were firmly crusted over.

The general condition of the patient was very poor during the first forty-eight hours of hospitalization. His temperature was only slightly elevated (99°F.). The pulse was somewhat rapid, varying in rate from 104 to 122

* I am indebted to Dr. C. H. Connor for permission to report this case.

per minute. Respiratory rate was 20 per minute on the first day. The blood pressure was not taken because of burned areas of the extremities. Nausea followed by emesis of 300 c.c. of dark brown fluid occurred in the late afternoon of the first day. Nausea continued and some abdominal distress was complained of. The urinary output during the first twenty-four hours was 75 c.c., this amount being obtained by catheter. The fluid intake had been 4,000 c.c. during this period. Local treatment was continued and general supportive measures, including subcutaneous fluids, morphine sulfate and digifolin, were administered.

During the early morning of July 13 (the second hospital day) the temperature rose to 103.8°F., with a pulse rate of 106 to 130. Involuntary urination occurred several times. Nausea persisted through most of the day, but no emesis occurred and ingestion of fluids by mouth was begun. The fluid intake was only 2,200 c.c. for twenty-four hours, but the output was 700 c.c. The urine was acid in reaction, had a specific gravity of 1.022, and numerous hyaline casts were present. Digifolin and morphine sulfate were given in therapeutic doses.

Improvement in general condition was noted on the third day. The temperature range was 98 to 101 degrees; pulse rate varied from 110 to 128. Abdominal distress was complained of early in the day and towards evening the patient became nauseated. The twenty-four-hour fluid intake was 3,500 c.c. The output of urine was 1,100 c.c., and contained many hyaline casts. The patient continued to take fluids by mouth.

On July 15 (the fourth hospital day), emesis of 500 c.c. followed the ingestion of some gruel and at 2:00 P.M. the patient vomited 100 c.c. of bright red blood. Fluids and nourishment by mouth were stopped. Five hours later considerable epigastric distress was complained of, but it subsided slowly. Digifolin was discontinued, morphine sulfate was given and an ice bag was placed over the epigastric region. The red blood count was 4,260,000; white blood count, 9,000; polys 70; monos 2; lympohcytes 28.

On the following day, emesis again occurred and the watery fluid was discolored by numerous dark flakes resembling old blood. Emesis of 200 c.c. of bright red blood, one large clot and several small clots, occurred on July 17 (the sixth hospital day). Some epigastric pain was present on the seventh hospital day. Hematemesis did not occur again while the patient was in the hospital and epigastric pain or distress was complained of on only two occasions thereafter.

As it became evident that bleeding had stopped, small frequent feedings of milk and cream were given and a régime of medical ulcer treatment with neutralizing powders of sodium bicarbonate with either calcium carbonate or magnesium oxide was ultimately established. This later included a soft bland diet.

The course of the patient's illness thereafter was essentially uneventful. Mild fever, presumably from the infected burned areas, and tachycardia persisted until August 24, 1934. The urine showed no further significant findings except a few coarsely granular casts on August 21, and a few renal epithelial cells on August 20 and September 3. Fluid intake and output were quite adequate and the patient was first permitted to be out of bed on August 12. Healing of the burned areas was progressing satisfactorily at that time and the infection was subsiding.

On August 31, 1934 a barium meal was given. The stomach appeared to be entirely normal. In the first part of the duodenum, seen under the fluoroscope, was a deformity on the lesser curvature which suggested duodenal ulcer. Films did not confirm this impression. The patient was discharged from the hospital on August 31, 1934.

Follow-Up Notes. The patient was next seen in February, 1937. He was well and was working regularly. A gain of about 25 pounds in weight was reported. In February, 1935, during the course of a respiratory infection, he experienced upper abdominal distress; he took some sodium bicarbonate and prompt relief was obtained. No other significant point was elicited. His appetite had been good, elimination was regular and there was no food intolerance.

On August 19, 1937 the patient was seen again. Mild distress before meals had been noted occasionally but was promptly relieved by food; some flatulence had been present from time to time. Food intolerance was denied. Under the fluoroscope the barium meal was observed to pass through the esophagus freely. The stomach filled readily, peristalsis was normal. The pylorus did not close to the tip due to a defect on the lesser curvature side. The base of the first portion of the duodenum

revealed defects on both sides. Irrespective of pressure, the defects remained constant during the fluoroscopy. Considerable tenderness was noted upon palpation over the pylorus and duodenum. The roentgenograms were confirmatory to the fluoroscopic findings in the duodenum but did not demonstrate those on the gastric side of the pylorus.

SUMMARY

1. A case of gastrointestinal ulceration following burns is reported.

2. No history of any gastrointestinal disturbance prior to the occurrence of the accident could be obtained. Emesis of bright red blood occurred on the fourth and sixth days after the burn. The patient was treated for ulcer thereafter by diet and neutralizing powders.

3. During convalescence, fluoroscopic examination by means of a barium meal yielded evidence of a duodenal deformity, but the films did not confirm this impression. These studies were repeated recently, three years after initial hematemesis, and evidence of both duodenal and gastric ulceration was demonstrated fluoroscopically. The films taken immediately thereafter recorded only the duodenal findings.

4. The discussion includes some points of historical interest, a consideration of the incidence and some aspects of the etiology, diagnosis and treatment of gastrointestinal ulceration following burns.

REFERENCES

1. DUPUYTREN, BARON. Clinical Lectures on Surgery, 1832. Translated from French by A. S. Loan. Boston, 1833. Carter Hendee Co.
2. CURLING, T. B. On acute ulceration of the duodenum in cases of burns. Med.-chir. Tr., 25: 260–281, 1842.
3. LONG, J. On the post-mortem appearances found after burns. London Med. Gaz., 1: 743–750, 1839–1840.
4. MOYNIHAN, B. G. A. Duodenal Ulcer, pp. 27, 28, 31, 38. Philadelphia, 1910. W. B. Saunders Co.
5. NOVAK, E. Gastro-intestinal ulceration following cutaneous burns; with report of case. Am. J. M. Sc., 169: 119–125, 1925.
6. LEONARD, V. N., and DAYTON, A. B. Multiple acute gastric ulcers after using Percy's "cold iron" for inoperable carcinoma. J. A. M. A., 66: 1549 (May 13) 1916.
7. PERRY, E. C., and SHAW, L. E. On diseases of the duodenum. Guy's Hosp. Rep., 50: 171–308, 1894.
8. RONCHESE, F. Duodenal ulcer after burns. Abstract in J. A. M. A., 83: 1038 (Sept. 27) 1924.
9. BANCROFT, F. W., and ROGERS, C. S. The treatment of cutaneous burns. Ann. Surg., 84: 1–18. 1926.
10. LEVIN, J. J. Duodenal ulcers following burns; with report of two cases. Brit. J. Surg., 17: 110–113 (July) 1929.
11. FENWICK. Quoted by Stengel in Modern Medicine, Osler and McCrae, Vol. 3, page 349, 1913.
12. HARRIS. R. I. Fatal burn: death due to hemorrhage into the suprarenal capsule and to hemorrhage from a duodenal ulcer. Brit. J. Surg., 16: 677–680 (April) 1929.
13. MAES, U. Curling's ulcer, duodenal ulcer following superficial burns. Ann. Surg., 91: 527–532 (April) 1930.
14. HUNTER, W. Pathology of duodenitis. Tr. Path. Soc. London, 41: 105–111, 1890.
15. FENWICK, W. S. Pathology of acute perforating ulcer of the stomach in young adults. J. Path. & Bact., 1: 417, 1893.
16. KAPSINOW, R. Mechanism of production of Curling's ulcer. South. M. J., 27: 500–503 (June) 1934.
17. WEISKOTTEN, H. G. Fatal superficial burns and the suprarenals. J. A. M. A., 69: 776 (Sept. 8) 1917.
18. MANN, F. C. A study of gastric ulcers following removal of the adrenals. J. Exper. Med., 23: 203–210, 1916.
19. ELLIOTT, T. R. Some results of excision of adrenal glands. J. Physiol., 49: 38–53, 1914.
20. LEVY, M., and ARMINGAT, J. Gastric ulcers produced by extirpation of the adrenals. Arch. d. mal. de l'app. digestif, 20: 1104–1108 (Nov.) 1930.
21. FREUD, J., LUWISCH, D., and OESTREICHER, F. Ulcerations in the stomach after adrenalectomy. Brit. M. J., 1: 1216–1217 (June) 1935.
22. McLAUGHLIN, C. W. The Curling ulcer. Study of intestinal ulceration associated with adrenal damage. Arch. Surg., 27: 490–505 (Sept.) 1933.
23. McLAUGHLIN, C. W. Intestinal ulceration following adrenal damage; experimental observations. Ann. Surg., 101: 554–558 (Jan.) 1935.
24. VON ROKITANSKY, C. F. Pathological Anatomy, p. 35. London, 1859. The Sydenham Society.
25. CUSHING, H. C. Peptic ulcers and the interbrain. Surg., Gynec. & Obst., 55: 1–34 (July) 1932.
26. MASTEN, M. G., and BUNTS, R. C. Neurogenic erosions and perforations of the stomach and esophagus in cerebral lesions. Arch. Int. Med., 54: 916–930 (Dec.) 1934.
27. GRANT, F. C. Brain lesions and duodenal ulcer; report of two cases. Ann. Surg., 101: 156–166 (Jan.) 1935.
28. WATTS, J. W., and FULTON, J. F. Effect of lesions of the hypothalamus upon the gastrointestinal tract and heart in monkeys. Ann. Surg., 101: 363–372 (Jan.) 1935.
29. HOFF, E. C., and SHEEHAN, D. Experimental gastric erosions following hypothalamic lesions in monkeys. Am. J. Path., 11: 789–802 (Sept.) 1935.
30. IVY, A. C. Contributions to the physiology of the stomach. LII. Studies on gastric ulcer. Arch. Int. Med., 25: 6–31 (Jan.) 1920.

31. KELLER, A. D. Ulceration in the digestive tract of the dog following intracranial procedures. *Arch. Path.*, 21: 127–184 (Feb.) 1936.

32. KELLER, A. D., and D'AMOUR, M. C. Ulceration in the digestive tract of the dog following hypophysectomy. *Arch. Path.*, 21: 185–201 (Feb.) 1936.

33. MATSUKUMA, M. Development of peptic ulcer following injury of midbrain in animals whose vagus nerve, celiac plexus, and celiac ganglion had been previously excluded. *J. Kumamato M. Soc.*, 13: 593–594 (Mar. 20) 1937.

34. BARDEEN, C. R. On certain visceral pathological alterations, the result of superficial burns. *Bull. Johns Hopkins Hosp.*, 8: 81–82, 1897.

35. BARDEEN, C. R. A review of the pathology of superficial burns, with a contribution to our knowledge of the pathological changes in the organs in cases of rapidly fatal burns. *Johns Hopkins Hosp. Rep.*, 7: 137–179, 1896.

36. PACK, G. T. Pathology of burns. *Arch. Path.*, 1: 767–805, 1926.

37. GLOBUS, J. H., and BENDER, M. B. Disseminated toxic degenerative encephalopathy (disseminated sclerosing demyelination) secondary to extensive and severe burns. *J. Nerv. & Ment. Dis.*, 83: 518–529 (May) 1936.

38. KEELEY, J. L., GIBSON, J. G. 2nd, and PIJOAN, M. Effect of thermal trauma on blood volume, serum protein, and certain blood electrolytes. In press.

39. WILBUR, D. L., and OCHSNER, H. C. Association of polycythemia vera and peptic ulcer. *Proc. Staff Meet. Mayo Clin.*, 10: 166–168 (Mar. 13) 1935.

40. SWINGLE, W. W., VARS, H. M., and PARKINS, W. M. Study of blood volume in adrenalectomized dogs. *Am. J. Physiol.*, 109: 488–501 (Sept.) 1934.

41. HARTZ, A. F., and DIGBY, W. E. Case of recovery from a duodenal ulcer produced by a burn. *Guy's Hosp. Rep.*, 52 (series III): 157–160, 1913.

42. ASHBY, J. E. Duodenal ulcer complicating burns in infant. *M. J. Australia*, 1: 183 (Mar. 3) 1917.

43. KIRCHMAYR, L. Duodenal ulcer after burns. Abstract in *J. A. M. A.*, 79: 690 (Aug. 19) 1922.

44. Choyce's System of Surgery, 2: 373–374, 1927.

45. SIMPSON, J. C. Case of extensive burns of thighs and abdomen. Probable ulceration of intestines. Recovery. *Brit. M. J.*, 2: 875–877, 1889.

46. FITZGIBBON, J. H. Duodenal ulcer following skin burns; report of two cases with recovery. *Northwest. Med.*, 31: 427–430 (Sept.) 1932.

47. MILLER, T., and RUBENSTEIN, B. Curling ulcer. *Dallas M. J.*, 18: 8–9 (Jan.) 1932.

48. CLEMENTS, H. J. Are we up to date in our treatment of burns. *Northwest. Med.*, 31: 209–215, 1932.

49. LAIRD, W. R., and WILKERSON, W. V. Curling's ulcer; with report of case. *West Virginia M. J.*, 27: 128–130 (March) 1931.

50. SWAIN. Extensive burn; hematemesis; death; necropsy. *Lancet*, 2: 1127, 1893.

51. D'EWART, J. Gastrostaxis following burns. *Brit. M. J.*, 1: 242, 1930.

52. WEINER, D. O., ROWLETTE, A. P., and ELMAN, R. Significance of loss of serum protein in therapy of severe burns. *Proc. Soc. Exper. Biol. & Med.*, 34: 484–486 (May) 1936.

53. ALDRICH, R. H. Role of infection in burns. The theory and treatment with special reference to gentian violet. *New England J. Med.*, 208: 299–309 (Feb. 9) 1933.

SURGERY OF THE CLEFT PALATE*

ARTHUR E. SMITH, M.D., D.D.S.   AND   JAMES B. JOHNSON, M.D.

Consultant in Reconstructive Surgery,   Attending Reconstructive Surgeon,
Veterans' Hospital   California Babies Hospital

LOS ANGELES, CALIFORNIA

CONGENITAL cleft palate is a condition due not to the thwarted development of the component structures that combine to form the normal palate, but to the failure of fusion of these parts in one or more locations, usually on either side of the midline. Of the congenital facial anomalies, none has commanded more attention than cleft palate and probably none has proved more difficult or baffling from the surgical standpoint.

It is impossible adequately to emphasize the mental hazards that have to be overcome by the unoperated or unsuccessfully treated cleft palate patient. The responsibility of the trained surgeon in restoring these unfortunate people to normal appearance and normal function in order to give them a chance of happiness and success in life, should not rest lightly upon his shoulders. The deformity should be corrected early in life because there is usually a gradual development of an endless chain of physical and mental sequelae.

It has been the belief and contention that the real foundation upon which an inferiority complex is based actually has its beginning during infancy and childhood, especially after the beginning of school life. At this time, the association with other children makes the unfortunate one cognizant of his deformity, regardless of the care exerted in the home to avoid this feeling of uneasiness. It is not uncommon for the parents of the unfortunate child to feel that they are responsible for nature's blunder and their feeling of sorrow and worry greatly accentuates the mental condition of the child. Remarks are made from time to time in the presence of these children which soon creates an influence of mental depression. As years pass, the cumulative effect of these factors produces a condition which is impossible to overcome completely even after successful reconstructive surgery has been performed. The victims of such a tragedy may remain wholly or partially secluded from society. They are not only seriously handicapped in securing an education, but find they have little or no chance in the business world. This is in no small measure due to the inferior personality which they have developed in addition to the actual deformity. It is only in very rare instances that these individuals gain promotions in the competitive fields of endeavor. Unless proper surgical reconstruction has been accomplished before the school age, additional difficulties are encountered in the classroom between the teacher and the pupil, due in most instances to the poor enunciation of the student or to the defects in hearing which are so commonly associated with this condition.

Throughout the past century, the literature has abounded with material on this subject and even as early as the time of Galen[1] (131–202) mention was made of cleft palate. Reports of the early observers indicate that surgery of one kind or another was attempted but reports of the end results in most instances are not at all clear. The earlier operators report some success in closure of the cleft, sometimes with residual patencies, but very little mention is made of the improvement of the speech mechanism, even though occasionally plans were offered whereby some improvement might be expected. During the past one hundred years the surgical

treatment has undergone a gradual evolution and many of the surgical procedures proved successful and adequate for closure

FIG. 1. Case 1. Female, age 21. Type 2 cleft. No history or previous operations. It was very difficult to understand her speech before operation.

FIG. 2. Same case. Note the long palate secured from the "push-back" surgical technique. After receiving adequate voice training, her speech has become normal. Operation done under block anesthesia.

of the cleft. The functional results associated with the speech mechanism of the soft palate were, however, still lacking.

We are of the opinion that surgical failure has been more common from cleft palate and cleft lip than any other type of congenital deformity. This is due perhaps to the fact that the surgery is often attempted by those unequipped for this difficult phase of reconstructive surgery. The rebuilding of a hard palate, with separated alveolar ridges and protruding premaxillary bone, in such a manner that a long flexible velum is obtained, requires not only surgical but mechanical skill and attentive postoperative care. It is not at all uncommon for the reconstructive surgeon of today to encounter patients who have had from two to ten operations and yet have a partially or completely open hard or soft palate with impaired speech. Since it is quite possible to correct these deformities, improper surgical technique must be blamed.

In outlining the treatment for any condition, a definite objective must be established. The surgeon must have in mind the removal of the many unusual aspects which combine to form this handicap. The cleft in the hard palate is not the most

important contributing factor, although its presence allows excursion of foods and liquids from the mouth into the nose and otherwise contributes to poor sanitation. It is only optimism or ego on the part of the surgeon to feel that satisfactory results have been obtained when merely a closure of the cleft has been accomplished. The nasal asymmetry with collapse of the tip cartilages on one side which is seen in such a large percentage of these cases constitutes another major problem for correction. With an associated cleft lip further to complicate the condition, the nourishment of the child becomes a definite problem. If this is not repaired early malnutrition or other conditions may result from diet deficiencies.

The chief factor concerned in removal of the handicap of cleft palate patients and the one which is most often neglected is the restoration of normal speech. Even though the cleft in the hard palate is closed and all external evidence of deformity is removed, the stigma has been diminished slightly or not at all if the speech remains grossly defective. This speech defect has a tendency to produce an inhibition towards speaking for fear that the condition will be exposed, especially since the patients feel it cannot be detected otherwise. In order to produce functional results in speech mecha-

nism, it is necessary that the palate be of sufficient length and the velum free from adhesions and tension. Although this is a

Fig. 3. Case ii. Male, age 18. Type 3 cleft. Two operations from 6 months to 2 years of age had been failures. Speech bad.

very difficult task, it is entirely possible and it is not at all uncommon where the operation is performed by a skilled reconstructive surgeon.

ETIOLOGY

In spite of the numerous theories, the exact etiology of cleft palate and lip is unknown. Heredity was mentioned as early as 1690 by Regis,[2] who considered heredity a very important predisposing factor in all developmental deformities. There is disagreement among numerous investigators regarding this theory.

Goeffrey St. Hilaire[3] thought that mechanical conditions were influential in bringing about the deformity. Vrolik and Nicati[4] suggested that a mechanical condition was produced by a hypertrophied tongue in cases of cleft palate which prevented union of the palatal plates. To this mechanical theory, many authorities agree.

Some of the other explanations of this condition are listed as follows: (1) disturbance in the pituitary gland; (2) altered function of the endocrines; (3) discrepancies between ages of father and mother; (4) in-and-in breeding; (5) changes in the

Fig. 4. Same case. Surgery completed. Note length of velum. Speech practically normal. Operation done under block anesthesia.

amniotic fluid; and (6) adenoids. These represent only a few of the theories and explanations offered for the etiology; it is beyond the scope of this paper to attempt to mention all. Practically every theory that has been proposed has received much opposition and now we find ourselves just as much at a loss to explain the true etiology of cleft palate and lip as were the early investigators.

INCIDENCE

Davis[5] has reported, after studying a large series of deliveries, that in white children there is one cleft per 1,000 births. In colored children, he found one cleft in 1,800 births. Although other reports do not coincide with his findings, the number of cases studied was considerably smaller and therefore not conclusive.

The data published on the various types of clefts are confusing although there is a marked similarity in the percentages. Since there has been no uniform method of reporting these statistics, it is difficult to understand the exact meaning of every report. We encounter some discrepancies which may or may not have significance. Table 1 summarizes some recent statistics.

We may draw the following conclusions: (1) the occurrence is relatively high in boys

as compared to girls; (2) left unilateral L.J.P. splits seem to occur about twice as often as right unilateral L.J.P.; (3) bilateral

FIG. 5 Case III. Male, age 21. Type 3 cleft. No history of previous operations. Speech very difficult to understand.

L.J.P. split is probably more common than right unilateral L.J.P. split; (4) cleft palate is more common in white children than in colored.

CLASSIFICATION

There is no universal classification for cleft palate. Some classifications are simple, treating the condition only in a general

with it. In order to expedite familiarity it must be rather simple but comprehensive. A classification which we evolved is based

FIG. 6. Same case. Note extreme length of velum and restoration of uvula. Speech practically normal. Operation done under block anesthesia.

on the anatomy of the deformity. In order to simplify our records, we have adopted the policy of referring to the type numbers rather than the description of the deformity. Since Type 1 begins at the uvula and the numbers progress as the cleft extends

TABLE I
OCCURRENCE OF VARIOUS TYPES OF CLEFTS
(All figures are percentages)

Author	Males	Females	Soft Palate	Hard Palate	L.U.L.J.P.*	R.U.L.J.P.	B.L.J.P.
Ladd[6]	....	....	....	....	55 0	29.0	16 0
W. B. Davis[7]	56.0	44.0	8 0	2 11	31 05	6.11	12.94
Lyons[8]	52.6	47 4					
Veau[9]	....	....	20.8	30 8	38 8		9 6
Authors	60.0	40 0	6 0	31 7	42 0	4.0	16 3

* Left unilateral.

way; others are very complicated and include every possible variety of associated cleft lip.

Unless there is universal adoption, a classification is no better than your familiarity

further forward, this classification becomes simple. No distinction is made between the sides on which the cleft occurs. This information can be added to the record.

This classification does not include every possible variation in cleft palate and no attempt has been made to include cleft lip except those associated with cleft palate.

Reference is made to our method of classification merely to state that we have found it most valuable in our office and for correct records.

Type I. Unilateral cleft of uvula.
    II. Unilateral cleft of soft palate.
    III. Unilateral cleft of soft and hard palate, but not including alveolar ridge.
    IV. Unilateral cleft of soft and hard palate including alveolar ridge.
    V. Unilateral cleft of soft and hard palate including alveolar ridge and associated with unilateral cleft lip.
    VI. Bilateral complete cleft and associated with bilateral cleft lip.
    VII. Median cleft.

CLOSURE OF CLEFT BY MEANS OF PROSTHETIC APPLIANCES

It is interesting to note from early records the many methods employed for mechanical closure of the open palate. The materials used have been sponges, cotton, wax, gum, gauze, silver plug or plate, gold plate, soft rubber, vulcanite and many others.

Much credit is due the pioneers for their ingenious mechanical contrivances in an attempt to improve the speech of the cleft palate individual. Since the advent of efficient impressions and models, many ingenious obturators have been designed, some of which have proved absolutely worthless while others have rendered only a limited service. These obturators were made for two purposes: first, to improve the voice and second, to prevent food from entering the nose.

The mechanical closure, regardless of its composition or its adaptability to the tissues, can only be compared to an artificial limb for comfort and service. A long flexible, vibrating velum is necessary for correct speech. It is therefore impossible for a rigid mechanical obturator to fulfill these important requisites because it does not vibrate and in addition it interferes with normal muscular accommodation. It also tends to produce irritation and is unhygienic.

We believe that a mechanical closure should only be used in the following cases: first, as a temporary measure, if necessary, to prepare for plastic reconstruction; second, advanced age; and third, advanced disease such as cancer, lupus, tuberculosis and syphilis.

The ideal method is the surgical closure of the open palate. This is made possible by utilizing the patient's own tissue from the oral cavity or tissue "waltzed" to the mouth by the "tubed" graft method.

SURGICAL TREATMENT

The Lip. Whenever cleft palate is accompanied by cleft lip, it is advantageous to repair the lip first. The ideal age for operation varies in different cases but in general it may be said that the lip should be repaired as soon after birth as possible. This can usually be accomplished during the first four weeks of life. Surgical intervention, however, is not advisable until the baby has been thoroughly examined and pronounced in good physical condition by the pediatrician.

Before attempting this lip closure, the position of the premaxilla must be determined. Abnormal protrusion of the premaxilla, if present, must be remedied before cheiloplasty is attempted. To reduce the protruding premaxilla, we employ the method of von Bardeleben which consists of a subperiosteal incision of the nasal septum followed by a gentle manipulation of the premaxilla. Care should be exercised to prevent overcorrection of the protrusion since the premaxillary tissues are normally located anterior, as pointed out by Blair, and not directly between the maxillae. The lip when repaired early exerts the continuous pressure upon the maxillae which diminishes the width of the cleft and

usually causes union in the region of the alveolar ridge. A much better result in the lip itself is accomplished by early surgery.

Lane also divided the normal lip in order to improve access to the mouth. Today, any procedure based on the eversion of flaps is

FIG. 7. Case IV. Male, age 26. Type 5 cleft. Cleft lip closed at age of 6 weeks. No history of cleft palate surgery. Speech extremely difficult to understand.

FIG. 8. Same case. Surgery completed. Note long velum and reconstructed uvula. Patient receiving voice training. Operation done under block anesthesia.

Before a result of the lip repair can be considered adequate, four requirements must be fulfilled: (1) The ends of the orbicularis oris muscles must be approximated; (2) the corresponding nostril must be restored to its normal contour; (3) the edges of the vermilion border must be accurately placed in proper alignment; (4) the scar line of the cleft must be reduced to a minimum. Unfortunately, too often one or more of these requirements are neglected by the untrained surgeon and everyone concerned is disappointed.

The repair of the palate is preferably postponed until the age of three or four years at which time the risk is considerably lessened and a narrowing of the cleft by nature has reached the maximum.

The Palate. Krimer,[10] in 1827, described a procedure for closure of a cleft by means of everting flaps of palatal mucoperiosteum. Davis-Colley,[11] in 1890, described a procedure based on the same principle, the difference being the location of the pedicle.

By 1897, Lane[12] modified the Davies-Colley operation and included mucous membrane from the soft palate in the flap.

usually spoken of as a Krimer-Lane operation. This method proved unsatisfactory in a large number of cases and today has practically been discarded.

Diffenbach,[13] in 1826, described a procedure for closure of clefts by means of mesial displacement of the palatal mucous membrane. Apparently this operation resulted in failure in practically 100 per cent of the cases.

Von Longenbeck,[14] forty years later, described his method of mesial displacement of the mucous membrane and incorporated with it the periosteum. In some cases he used one lateral incision, while in others two were necessary. The posterior palatine artery was left intact. This procedure became very popular and has remained so until the present day—many present day procedures consisting only of modifications of von Longenbeck's method.

Reeves,[15] a contemporary of von Longenbeck, suggested the correction of clefts in the palate by approximating the edges of the separated maxillae. He believed that the defect was due to a spreading of the maxillary bones.

This procedure was modified by Brophy who championed the forced compression technique and discarded all other procedures in cleft palate surgery. He recommended direct wiring of the bones in the midline after severe force had partially or completely approximated the edges. The wires were tightened from time to time until the desired result had been accomplished. Other methods for forced compression have been described which include pressure from outside the mouth by means of various appliances and headpieces, and pressure from within the oral cavity which usually consisted of some orthodontic or prosthetic appliance with adjustable screws fastened to the teeth for continuous force. Brophy[16] erroneously claimed that it was possible for the experienced surgeon to drill through the substance of the maxillae without disturbing the tooth germs. He also stated that the surgeon must be able to detect the feeling of a tooth when the instrument comes in contact with it and to change the course of the drill so that it passes between the teeth.

Brophy[17] made the following statement in 1907: "Having operated on over 1300 cleft palate patients, over 400 of whom were infants under 6 months old, I have found the breadth of the upper jaw as compared to the lower jaw is just as much broader than it should be as the distance between the borders of the fissure. When we bring the borders of the fissure into contact we have brought the upper jaw back to its normal breadth and to its proper relation with the lower jaw. There are very few exceptions to this statement."

Peyton[18] measured ninety-one normal infants and twenty-six with clefts that extended through the alveolar ridge. He also quotes an unpublished series of 600 by Richdorf, comparing the measurements of the normal palates to those containing clefts. Peyton found that the dimensions of the cleft palate, except surface width, were longer than those of the normal palate and that the width of the cleft diminishes 1.5 mm. during the growth interval between

51 and 59 cm. crown-heel length. These two factors contradict the statement made by Brophy and lead us to believe that forced compression is an unnecessary procedure. The severe force which is often necessary to produce a sudden approximation of the bone edges may cause fractures of other bones of the face, thereby producing an additional trauma and deformity.

From observation of a large number of patients in which forced compression was employed, we are led to agree with the adverse views expressed by G. V. I. Brown, Dorrance, John Staige Davis, Pitts, Veau, Stone, Blakeway and many others in condemning this procedure. We believe that the majority of cleft palate surgeons have discarded this method due to its injurious effects upon the tooth germs and the other tissues of the mouth. Another disadvantage is the failure to take into consideration the length and mobility of the velum which is insufficient in such a large percentage of cleft palate patients and which is so essential to the production of normal diction.

Surgical Technique for Producing a Long Velum. The "push back" operation was originated by Dorrance[19] and is performed in two stages. The object of the first stage is to establish collateral circulation and the second to effect closure of the cleft.

Lateral incisions through the mucoperiosteum are made on either side 2 or 3 mm. from the lingual gingival margin of the teeth and extending from the region of the lateral incisors anteriorly to the tuberosities posteriorly. The mucoperiosteum is separated from the bony palate except anteriorly in the region of the central and lateral incisors. The posterior palatine arteries are severed and the nasopalatine arteries are left intact. The mucoperiosteum is now sutured into its original position.

Three to six weeks are allowed to elapse for good collateral circulation before attempting the second stage. The incisions for the second stage are in the same location as those of the first but are extended anteriorly to meet lingual to the central

incisors and posteriorly over the pterygo-mandibular folds. The mucoperiosteum is entirely separated from the bony palate

FIG. 9 Case V. Female, age 22. Type 3 cleft. Operation at 3 years had been failure. Speech very poor.

including its attachment at the posterior border. With a chisel, the hamular process is divided above the medial pterygoid plate. The mucoperiosteal flaps are now approximated in midline with the posterior margin of the velum in contact with the pharyngeal wall.

After the edges of the cleft are denuded, Dorrance inserts an aluminum-bronze suture into the gums which is passed from one side of the soft palate to the other and back again, the ends being twisted over one another to relieve tension on midline sutures. For retention of the mucoperiosteal flap against the bony palate, Dorrance passes sutures through the bone and in addition he advocates wires passed transversely around the bicuspid teeth and moulded around the dental arch meeting anterior to the incisor teeth where the wires are twisted together. An iodoform gauze pack is then placed between the wire splint and palate.

In cases of congenital shortening of the palate, we are using Dorrance's technique with certain modifications which we have found to be more satisfactory in our hands. For retention of the mucoperiosteal flap against the bony palate, we employ three strands of 26-gauge stainless steel wire

which are passed between the teeth across the palate and between similar teeth on opposite side, where they are twisted. This

FIG. 10. Same patient. Surgery completed. Note long velum. Speech normal. Operation done under block anesthesia.

is followed by the insertion of three rolls of sponge rubber packs which are placed between the wire and palatal flaps. The two lateral packs, which are smaller and inserted first, force the incised edges against the lingual plates of the bony vault and the larger central pack acts as a keystone and locks the two lateral packs in place.

We have adopted the policy of postponing the second operation for eight to twelve weeks to allow for ample collateral circulation.

In closing the edges of the denuded cleft, we are using three rows of sutures. The nasal mucous membrane is closed with interrupted silk. The muscles are approximated with triple o plain catgut and the oral mucous membrane is closed with Deknatel.

The aluminum-bronze wire as recommended by Dorrance for retention has proved unsatisfactory to us. We found it very difficult to insert and still more difficult to remove, consequently producing

unnecessary damage to the tissues of the velum. For retention, we are using with much satisfaction a ¼ inch lead band. The

using gauze packs saturated with surgical dressing in the relaxation incisions between the tuberosities and the lead plate. This

FIG. 11. Case VI. Male, age 22. Type 6 cleft. History of nine operations from 2 weeks to 14 years of age, all failures. Speech so bad that it was difficult to take history.
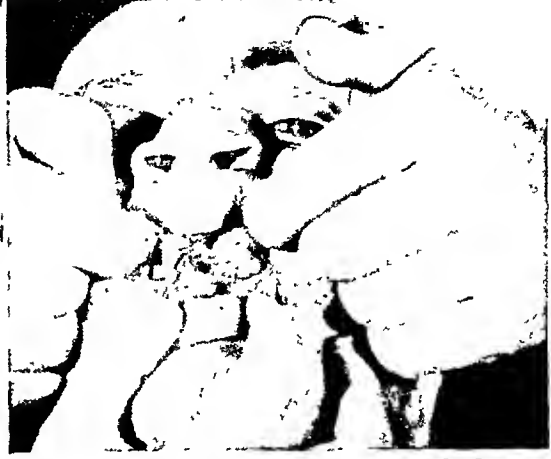
FIG. 12. Same patient Surgery completed. Normal speech obtained. Operation done under block anesthesia.

correct length of the lead band is first ascertained by passing a suture around the tissues to be held by the band. The prepared lead band is passed around the loosened palatal tissues so that the ends approximate in the midline on the oral side of the palate. To insure stability, the band is slotted in eight places, four on each side, that is, two anteriorly and two posteriorly. Two small holes are drilled in the ends of the band and the passage of sutures holds the ends in approximation with each other. The appliance is now adjusted to position and the ends approximated and tied. Interrupted sutures of Deknatel are passed through and through the palate at the location of each slot on the lead band and tied on the oral side of the palate. We believe that this band produces a maximum of retention with a minimum amount of trauma.

We have adopted the use of rubber sponge packs in preference to the iodoform gauze because we feel that the continued resiliency of the rubber is superior to the gauze packs. We also feel that by using three individual packs instead of one, the flap is held in place by more uniform pressure throughout its length. We are also

prevents early contraction in this region and also aids in decreasing the tension on the suture line. These packs are removed on the sixth day. For the closure of a Type IV or V in which the alveolar ridge is involved, we are using two Y-shaped flaps which extend over the alveolar ridge. These flaps are raised during the preliminary operation and are included with the mucoperiosteal flaps of the hard palate. When the palatal tissues are pushed back, the Y-shaped flaps are closed by sutures in the median line. In this way, patencies in the region posterior to the incisors are prevented, thus eliminating the use of prosthetic appliances. The denuded area here, as in other areas of the hard palate, regenerates by granulation. We have obtained excellent results with this procedure.

VOICE TRAINING FOLLOWING CLEFT PALATE SURGERY

Relation between the Length of the Velum and Normal Speech. Most important among the contributing factors to personality is the voice. Personality is reflected in vocal tone, the quality of which sends out vibrations musical or otherwise.

The speech difficulties which arise from cleft palate are of the mechanical nature

the difficulties involved sometimes depending upon the type of cleft. The inability to make explosive stops ranging from the palate and at times including the lips is universal with this condition. Such stops are made in the consonants *g, k, d, t, b, v* and *p*. Discrepancies in the anatomic structures produce a condition in which the fricative consonant is difficult because the breath cannot be concentrated to form whistling sounds in *f, s* or *z*. The majority of cleft palate individuals have spoken so long without full benefit of their vocal cords that there is a tendency for the cords to lose their tone. Abnormal breathing and posture are generally associated with cleft palate and are closely related to normal voice projection and therefore should be corrected.

If the surgery is not completed between the ages of 3 and 4 years, the problem of mental development is intensified with the succeeding years. Since so many patients are not operated upon during childhood, it is advisable to give intensive voice training in order that these individuals may adjust themselves mentally and be prepared to enter competitive social and business relationships.

After the palate is reconstructed, the capable surgeon produces a closed palate and a long flexible soft palate in which the muscles are able to function normally after which there is no organic reason why the cleft palate speech should persist. Surgery which leaves the palate scarred, tight, contracted and too short, does not relieve the mechanical difficulties with consonant formulations and therefore cannot be considered adequate. However, in such cases, speech training will improve the enunciation. Whether or not the palate is adequately closed and especially after the patient has reached adulthood before surgery is attempted, it is necessary in most instances to rehabilitate the muscles concerned in velopharyngeal closure by means of some form of speech training. We feel that only a part of our professional service has been rendered when the surgery is completed, and that every patient should have the proper guidance necessary for a return to normal speech. We require all patients needing assistance to avail themselves of adequate speech training, which we have available in our office. We are employing a former cleft palate patient whose speech is so improved that it is impossible to detect that she has had a cleft palate. This patient received her training from Mme. Mae Albers, who is associated with us in this work. .

Unless the patient or the patient's parents agree to speech training, the individual is not accepted for reconstructive surgery. It is found that besides the desire for better appearance and improvement of oral and nasal hygiene, speech improvement is the hope of those seeking such reconstructive surgery.

CONCLUSIONS

1. Cleft palate is one of the more common congenital anomalies requiring careful and adequate surgical treatment.

2. Surgery that results in a short contracted velum must be considered a failure.

3. Judging from the patients we have observed, forced compression of the superior maxillae has produced a great deal of unnecessary damage in nearly all instances.

4. Cleft lip should be operated on as soon after birth as the physical condition will permit.

5. Cleft palate should be operated on between the ages of 3 and 4 years.

6. Prosthetic appliances may have their place in rare instances.

7. The "push back" operation when properly performed will produce a long flexible velum so necessary for normal speech.

8. Certain minor modifications which we have·adopted have increased the value of the "push back" operation in our hands.

9. In all patients, voice training is indispensable for the restoration of speech.

REFERENCES

1. GALEN. Cited by Dorrance. The Operative Story of Cleft Palate. Philadelphia, 1933. Saunders.

2. Regis, P. S. Cited by Dorrance.
3. St. Hilaire, Geoffrey. Cited by Dorrance.
4. Vrolik and Nicati. Cited by Dorrance.
5. Davis, J. S. Incidence of congenital clefts of the lip and palate. *Ann. Surg.*, 80: 363, 1924.
6. Ladd, W. E. Harelip and cleft palate. *Boston M. & S. J.*, 194: 1016, 1926.
7. Davis, W. B. Harelip and cleft palate. *Ann. Surg.*, 87: 536, 1928.
8. Lyons, C. J. Etiology of cleft palate and lip and some fundamental principles in operative procedure. *J. Am. Dent. A.*, 17: 827, 1930.
9. Veau, Victor, Division palatine. Paris, 1931. Masson et Cie.
10. Krimer, W. Division anormale du palais osseux guérie par la staphyloraphie. *Arch. gén. de méd.*, 18: 426, 1828.
11. Davies-Colley. On a method of closing cleft of hard palate by operation. *Brit. M. J.*, 2: 950, 1890.
12. Lane, Sir W. A. Cleft Palate. London, 1897.
13. Diffenbach, J. F. Ueber das Gaumensegel des Menschen und der Saengethiere. *Litt. ann. d. ges. Heilk.*, 4: 298, 1826.
14. von Longenbeck, Bernhard. On uranoplastics. *Med. Times Gaz.*, 2: 69, 1861.
15. Reeves. Cited by Blair. Surgery for Diseases of Mouth and Jaws. St. Louis, 1918. C. V. Mosby & Co.
16. Brophy, T. W. *J. A. M. A.*, 49: 662, 1907.
17. Brophy, T. W. *Boston M. & S. J.*, 2: 656, 1896.
18. Peyton, W. D. *Arch. Surg.*, 22: 661, 1931.
19. Dorrance, G. M. Congenital insufficiency of the palate, *Arch. Surg.*, 21: 185, 1930.

Any irritant if weakened sufficiently becomes a stimulant, and the farther a tissue is from the site of the irritant, the stronger will be the tendency to stimulation rather than destruction.

From—"A Textbook of Surgery," edited by Frederick Christopher (Saunders).

SURGICAL RELIEF OF PAIN DUE TO CIRCULATORY DISTURBANCES OF THE FEET*

REPORT OF A NEW METHOD

SAMUEL PERLOW, M.D.   AND   S. SHERMAN HALPERN, M.D.

Instructor in Surgery, Northwestern University
Medical School; Adjunct Attending Surgeon,
Michael Reese Hospital

Clinical Assistant in Surgery, Michael Reese
Hospital

CHICAGO, ILLINOIS

ONE of the greatest problems in the treatment of circulatory disturbances in the lower extremities is the relief of pain. In the mild cases rest and sedatives are usually sufficient until the circulation is improved by the various therapeutic measures. In the severe cases where ulcers and gangrene of the toes have occurred or where there is ischemic neuritis, more active measures are necessary.

Injection of alcohol into the tibial nerve for relief of this pain was carried out first by Silbert[1] in 1922. In 1928 Smithwick and White[2] recommended destruction of the various nerves to the feet by crushing or by alcohol injection through a small incision in the skin directly over the nerve desired. Since then a number of reports have appeared in the literature on the use of this procedure, with excellent results in a large series of cases. We wish to report our experiences with peripheral nerve anesthetization for the relief of pain in the feet of patients with peripheral circulatory disturbances, both by the operations recommended by Smithwick and White and by the intraneural injection of slowly absorbed anesthetic solutions.

In order to carry out either of the above procedures on the peripheral nerves of the feet a very detailed knowledge is required of the course of the various nerves and their relations to the surrounding tissues.

Nerves. There are five nerves which are sensory to the foot, i.e., the posterior tibial nerve which is sensory to the plantar surface of the foot and heel and to the dorsal surface of the distal phalanges; the sural nerve which is sensory to a triangular area on the lateral surface of the foot extending from the ankle towards the fifth toe; the saphenous nerve which is sensory to a similar area on the medial side of the foot; the superficial peroneal nerve which supplies the dorsum of the foot; and the deep peroneal nerve which is sensory to a triangular area between the first and second toes.

The posterior tibial nerve is the one which is most frequently treated for relief of pain. It divides under the laciniate ligament into the calcaneal, and the medial and lateral plantar nerves. It is about 6 mm. in diameter and is inclosed in a sheath with the posterior tibial vessels. In the distal third of the leg it lies lateral to the vessels between the flexor digitorum longus and the flexor hallucis longus muscles and on the tibialis posterior muscle.

The sural nerve is formed on the gastrocnemius tendon at the junction of the lower and middle thirds of the leg by anastomosis of the medial sural cutaneous nerve and the anastomosing peroneal branch of the lateral sural cutaneous nerve. It passes inferiorly with the lesser saphenous vein along the lateral edge of the gastrocnemius tendon and behind the lateral malleolus to its distribution on the lateral surface of the foot.

The saphenous nerve lies behind the greater saphenous vein superficially on the medial side of the leg.

The superficial peroneal nerve, after giving off its motor fibers high up, becomes entirely sensory at the junction of the the lower part of the leg it lies between the tibialis anterior muscle on its medial side and the extensor digitorum longus and the
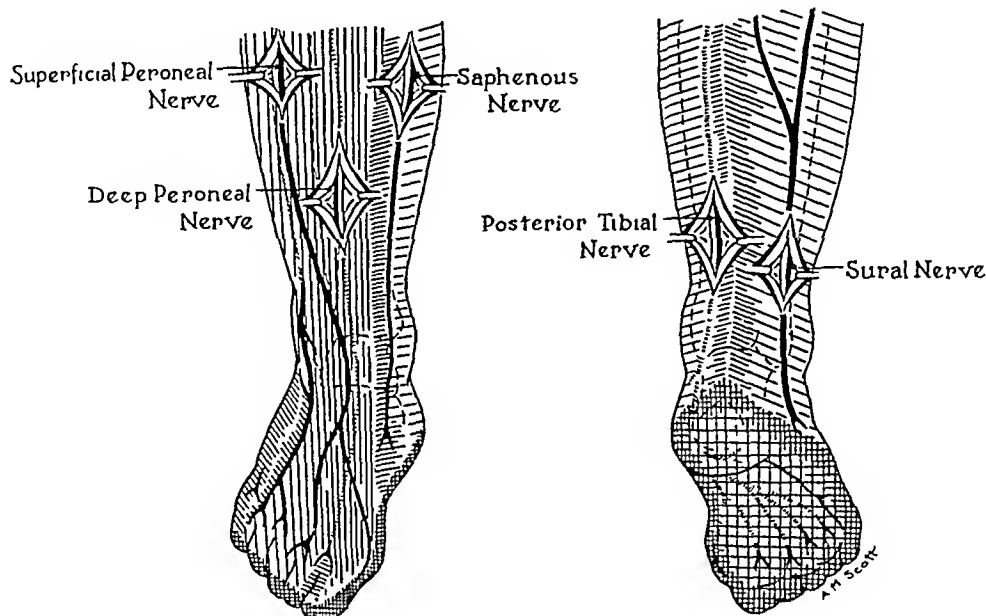
FIG. 1. Ideal sites for skin incisions for the various nerves. The solid line shows the course of the nerve in the subcutaneous tissue. The dotted line shows the course of the nerve under the fascia. The cross hatching shows the distribution of the nerves to the skin of the foot and leg.

lower and middle thirds of the leg. At this point the nerve trunk lies under the deep fascia on the extensor digitorum longus muscle about 3 cm. lateral to the crest of the tibia. Its course is somewhat in the medial direction. Half the nerve fibers pierce the fascia here and become the medial dorsal cutaneous nerve to the foot, which courses medially and anteriorly across the ankle to supply the skin of the medial half of the dorsum of the foot and the first, second, and part of the third toes. The remaining fibers of the superficial peroneal nerve pierce the fascia somewhat lower down, about 5 cm. above the lateral malleolus on the anterior surface of the leg, and become the intermediate dorsal cutaneous nerve to the foot. This passes anterior to the lateral malleolus, anastomoses with the sural nerve and supplies the lateral half of the dorsum of the foot and the third, the fourth, and the fifth toes.

The deep peroneal nerve is sensory to the skin between the first and second toes. In

extensor hallucis muscles laterally. Just above the ankle the nerve lies behind the tendon of the tibialis anterior and between

FIG. 2. Area of anesthesia produced by anesthetic in oil injection into posterior tibial nerve at site shown by dot. Arteriosclerotic ischemia with painful ulcer on big toe. Anesthesia lasted four weeks. Skin temperature rose 3.2°c. Ulcer healed.

the tibia medially and the extensor hallucis and the extensor digitorum muscles laterally.

Technique. The technique of alcohol nerve block, as described by Smithwick and White, is simple. Under local anesthesia

with 1 per cent procaine solution the nerve desired is isolated above the ankle and either crushed or cut. From ½ to 2 c.c. of 95 per cent alcohol is injected directly into the trunk. The nerve is then dropped back into place and the wound closed with fine

on the medial side of the leg at the junction of the middle and upper thirds. It will be found close to the greater saphenous vein. The superficial peroneal nerve can be isolated through an incision made 3 cm. lateral to the crest of the tibia

TABLE I
PERIPHERAL NERVE OPERATIONS

| Diagnosis | No. of Cases | Nerves | | | | | Incisions | | | | | Amputations |
| | | Posterior Tibial | Sural | Saphenous | Superficial Peroneal | Deep Peroneal | No. | Healed | | Drained | | |
								Alcohol	Crushed	Alcohol	Crushed	
Thrombo-angiitis obliterans	30	29*	8	6	9*	6	46	18	16	10	2	9
Arteriosclerosis	10	8	2	..	5	4	14	3	8	3	0	4
Polycythemia	1	1	..	..	..	..	1	1	..	0		
Total	41	38	10	6	14	10	61	22	24	13	2	13

* Two cases of thrombo-angiitis obliterans had second tibial and superficial peroneal nerve block when pain recurred one and two years later, respectively.

catgut or silk. Care must be taken to protect the surrounding tissues from the alcohol. Because of the poor healing power of the tissues with which one is dealing, it is absolutely essential to isolate the nerve trunk high up above the ankle and to work through a small incision (2 to 3 cm.) and with as little injury to the tissues as possible.

The posterior tibial nerve trunk is best isolated 5 to 10 cm. above the medial malleolus along the medial edge of the gastrocnemius tendon. This tendon and the belly of the flexor hallucis longus muscle should be retracted laterally and the tendon of the flexor digitorum longus muscle should be retracted medially. The sheath containing the nerve and the vessels will be found about 2 to 3 cm. deep, the vessels being medial and the nerve lateral. The sural nerve can be isolated through a small incision about 5 to 10 cm. above and behind the lateral malleolus along the lateral edge of the gastrocnemius tendon. It lies lateral and slightly anterior to the lesser saphenous vein. The saphenous nerve can be isolated through an incision

at the junction of the middle and lower thirds of the leg. The nerve will be found enclosed in a fascial compartment between the anterior tibial and peroneal groups of muscles. The deep peroneal nerve is isolated best through a vertical incision 10 cm. above the medial malleolus along the crest of the tibia. The tendon of the tibialis anterior muscle must be raised and retracted laterally. The nerve and the vessels will be found about 2 to 3 cm. deep, lying on the interosseous membrane close to the bone. The nerve lies anterior to the artery and vein.

When it is desired to block two nerves, as the superficial and deep peroneal nerves, or the superficial peroneal and the saphenous, or the posterior tibial and the sural nerves, it is not always necessary to make separate incisions in the skin for each nerve. By making the skin incision midway between the desired nerves and stretching the skin slightly both can be isolated through a single incision.

In the past few years we have performed a total of seventy-eight operative nerve blocks in forty-one patients. (Table I.)

Twenty-eight patients were able to continue with conservative treatment which ultimately resulted in establishment of an adequate circulation. In thirteen the gangrenous process continued, although the pain was relieved, and major or minor

TABLE II

NERVES INJECTED WITH ANESTHETIC IN OIL SOLUTIONS

	No. of Nerves	No. of Injections into Each Nerve	Total Injections
Posterior tibial.......	21	1–4	46
Sural...............	2	1–2	3
Common peroneal....	2	1–2	3
Superficial peroneal ..	2	1–2	3
Deep peroneal........	2	1–2	3
Saphenous..........	1	2	2
	33	...	60

amputations were necessary. Following the nerve block there was obtained in almost all our cases a rise in the skin temperature in the anesthetized area of 0.5° to 6°c., due to relief of the vasospasm. Because of this improvement in the circulation, in a number of our cases long standing ulcers healed promptly without further therapy.

The operation of nerve block is not without risk, however. Of the sixty-one separate incisions made in the skin to block the seventy-eight nerves, forty-six healed by primary union, thirteen drained for varying lengths of time and two failed to heal. In the latter two incisions large sloughing ulcers developed which were more painful than the original ischemic neuritis and necessitated amputation of the leg. One incision for the posterior tibial nerve broke down and an ulcer developed at its site two years after it had healed by primary union. Only one trophic ulcer developed in all of the patients operated on. That was in a laborer who used his anesthetized foot to apply pressure to his shovel in digging. The ulcer healed quickly when the foot

was placed at rest. All but two of the wounds that drained, occurred in cases in which alcohol was injected into the nerve, apparently due to tissue destruction by the alcohol. In the more recent cases, in which the nerves were simply crushed or cut and no alcohol was used, all the wounds healed by primary union except in the one patient who developed the large sloughing ulcers at the site of the 2 incisions. We believe now that in this latter case the local circulation was too poor to withstand any operative trauma.

Because of this unfortunate result and because there were other cases where severe pain of the feet was accompanied by poor circulation so that we hesitated to perform nerve block, we began a search for some other simple pain-relieving measure. We found that repeated infiltration of the various nerves with 1 per cent aqueous procaine solution gave relief for an hour or two in the severe cases and for as long as twenty-four hours in the mild cases of burning sensation in the soles. In several patients with continuous burning sensation in the soles of the feet we were able to give relief for relatively long periods of time by frequently repeated injections with aqueous procaine solutions. Apparently the fibrosis produced by these injections was sufficient partially to block the sensory fibers.

In order to obtain more prolonged anesthesia by single injections, we resorted to oil solutions of the anesthetic. Oil solutions are, being used extensively in relieving the postoperative discomfort of rectal cases and in pruritus ani and vulvae. Their use for relief of pain in peripheral circulatory disturbances by injection of the solution directly into the peripheral nerves has never been tried before, to our knowledge. We tried the following: procaine base 0.075 gm.; butesin 0.30 gm.; benzyl alcohol 0.25 gm.; oil of sweet almond sufficient to make 5 cc.* We have used this solution for twenty-

* Prepared by Abbott Laboratories under the name of butecaine.

nine nerves in seventeen cases. In each case complete anesthesia was produced for varying lengths of time in the area innervated by the nerve injected. Because of the difficulty in depositing enough oil within the nerve sheath, it was necessary to inject some of the smaller nerves several times before the anesthesia was produced. The anesthesia was accompanied by a rise in temperature in the same area which varied between 0.2° to 5°c. The duration of anesthesia produced varied from one to forty days. Most of the nerves had to be reinjected to relieve the pain before sufficient collateral arterial channels were established by the conservative treatment used.

One of the dangers of injection of oily solutions anywhere is suppuration and sloughing of the skin when the solution is injected intradermally. This possibility must always be kept in mind, especially when large quantities of the solution are used. We were able to avoid this by using only small quantities (1 to 2 c.c.) and injecting directly into the nerve. In several instances the site of the nerve injection remained tender for several days after the solution was introduced, but no treatment was necessary for this. There was no suppuration at any time.

When the procaine in oil solution was injected into a mixed nerve, the motor fibers, as well as the sympathetic and sensory, were anesthetized. In the case of the posterior tibial nerve the small plantar muscles which are of only slight use in walking are paralyzed. To prevent their atrophy and the resultant weakness of the longitudinal plantar arch, the muscles were massaged daily and leather arch supports applied. In the case of the common peroneal nerve the resultant paralysis involved all of the anterior group muscles and caused a foot drop. For this reason this nerve was injected only when the superficial branch could not be located. We have discontinued the injection of this nerve and now prefer to crush the superficial peroneal nerve

through a high incision when we cannot locate it with a needle.

Technique of Injection. The oil solution is drawn up into a small syringe and a 1½ inch 23 gauge needle is attached. The skin over the nerve is painted with iodine and alcohol, and the needle is inserted to the location of the nerve. This will be evidenced when the patient indicates the occurrence of a sharp pain in the area of nerve distribution. The solution should then be injected very slowly. One must be certain that none of the solution is injected into the skin or into a blood vessel. The nerves are located in the same regions as those used for operative blocking. (Fig. 1.)

SUMMARY

Operative peripheral nerve block by alcohol or crushing was performed for relief of pain in peripheral circulatory diseases of the feet seventy-eight times, through sixty-one incisions in forty-one cases. Forty-six of the incisions healed by primary union, thirteen drained for varying lengths of time and two developed large sloughing ulcers. Twenty-eight of the patients so treated were able to continue with conservative treatment and ultimately developed a good collateral circulation. In thirteen cases the gangrenous process continued in spite of the relief from pain and ultimately resulted in amputation. This high percentage of amputations was due to the fact that most of the patients treated by nerve block already had extremely poor circulation, gangrene, ulcers or infections.

Procaine in oil solutions were injected into thirty-three peripheral nerves of twenty-one patients suffering from pain in the feet due to peripheral circulatory disturbances. This anesthetized the sympathetic, sensory and motor fibers. The anesthesia and the rise in temperature due to relief of the vasospasm lasted from one to forty days. The motor paralysis that resulted when motor fibers were contained in the anesthetized nerve did

not last as long as the sensory anesthesia. No harmful results were produced by the procaine on oil injection into the nerve.

We have found peripheral nerve injection with procaine in oil to be a valuable adjunct in the treatment of pain due to peripheral circulatory disturbances of the feet. The anesthesia produced is similar to that obtained by peripheral nerve block with alcohol or crushing but is not so long lasting. However, it can be repeated as desired and has the advantage of not requiring an operative incision.

REFERENCES

1. SILBERT, S. Treatment of thrombo-angiitis obliterans. *J. A. M. A.*, 79: 1766 (Nov. 18) 1922.
2. SMITHWICK, R. H., and WHITE, J. C. Elimination of pain in obliterative vascular disease of the lower extremity. *Surg., Gynec. & Obst.*, 51: 394–403, Sept. 1930.

THE bite of a black widow spider may be soon followed by severe abdominal pain; examination may reveal a hard "board-like" abdomen which simulates that observed in perforated peptic ulcer. There is, however, little associated tenderness and within a few hours the signs and symptoms gradually disappear although distention may be present and persist for several days.

From—"Textbook of General Surgery," by Warren H. Cole and Robert Elman (D. Appleton-Century).

STUDIES REGARDING SILK AND CATGUT IN INVAGINATION OF THE APPENDICIAL STUMP; AND REGARDING NON-INVAGINATION TECHNIQUE*

J. K. DONALDSON, M.D.   AND   HARVEY S. THATCHER, M.D.

Associate Professor of Surgery, University   Late Professor of Pathology, University
of Arkansas School of Medicine   of Arkansas School of Medicine

LITTLE ROCK, ARKANSAS

IN a recent presentation[1] we discussed a series of experiments in which we compared the so-called ligation and drop method or non-invagination technique of doing appendectomies with the invagination or purse-string technique. In this previous study silk was used for invaginating the appendicial stump. In this present article, we discuss further studies which have been made comparing a group in which catgut was used for invagination. In the previous report, tables showing results of microscopic study of the buried stump regions were correlated with gross adhesion readings; and a short review of literature upon the subject was presented. In this present report unnecessary repetition of review and of tables is avoided, those interested in additional detail being referred to previous articles upon the subject which are listed in the accompanying bibliography. For the sake of completeness, however, in presenting the conclusions and comparisons of this present study certain previously mentioned points must be briefly discussed.

One of us adopted the non-invagination method about nine years ago, after becoming convinced that the reasoning of such men as the Horsleys, Baldwin, Charles Mayo and others was correct in that the non-invagination method could be more quickly executed, would decrease the danger of abscess formation by avoiding submergence of a contaminated stump in a blind pocket, as well as reduce the incidence of postoperative fistula, adhesion formation and intestinal obstruction. In the light, however, of continued experimental and clinical experience within the abdomen in procedures dealing with adhesion formation and others, we came not long ago to question the logic of the non-invagination technique.

A review of the literature comparing the two techniques under discussion impresses one with the following points: (1) Practically all the clinicians who have supported either the non-invagination technique or the inversion or invagination techniques have based their arguments on purely clinical observations. (2) Complications are so rare in appendectomies done on uncomplicated cases by either method under discussion, even in the experience of very busy surgeons, that it seems probable that conclusions drawn from purely clinical observations cannot be thoroughly relied upon.[1] (3) As many or even more prominent clinicians have advocated the invagination technique as have supported the non-invagination technique. (4) Experimental work upon the subject is limited. (5) Practically all who have done experimental work on this problem have come to the conclusion that the invagination technique is better.

Though one must admit that clinical complications due to either method per se are rare, nevertheless the highest degree of perfection obtainable in surgery is certainly desirable. And if the incidence of postoperative intestinal obstruction from abdominal adhesions might be lowered, additional experimental and clinical study will have been worthwhile.

METHOD OF EXPERIMENTATION

Dogs were used in these experiments. In the dog the appendix frequently resembles very closely that found in the human. It also, however, is often similar to a true diverticulum. On executing our experimental procedures we felt that it would be essential that we avoid the routine dissection of the appendix to its base. If dissection allowing a complete freeing of all the appendix is carried out routinely, one will frequently encounter bleeding more difficult to control than in the human. One will also expose a variable amount of raw surface, making the procedure so variable that one could not later have consistent readings regarding adhesion formation.

Consequently, we adopted the routine of avoiding altogether dissection of the appendix from its bed. In many instances the entire appendix could be removed without this dissection, a ligature being properly applied to the base and the stump buried. (Figs. 1 and 2.) With a broader and larger appendix we simply removed the free tip and invaginated the ligated stump within the lumen of the appendix itself. (Fig. 3.) When practical, we invaginated the entire large base and stump into the bowel lumen. We feel that we were able to a great extent to avoid by the technique used variable factors which might confuse or distort our readings.

We removed the appendix in some with the actual cautery and in others with the cold knife, applying carbolic acid and alcohol to the stump. No detailed comparative studies were made regarding the cold knife and actual cautery method. For the purpose of this experiment, we believe one method proved as good as the other.

Davis and Geck chromic O, non-boilable, twenty day catgut on an intestinal needle, the type of purse-string suture commonly used in this country when catgut is used for burying the stump, was utilized as a purse-string in the catgut group.* Ordinary

* We wish to thank Dr. Clock of the Davis and Geck Company for furnishing us with suture material used in this and other experiments.

sewing silk purchased at a clothing store, equivalent to about a size 1 surgical silk was utilized as a purse-string when silk was used. When the stumps were not buried, they were sometimes singly and sometimes doubly ligated with chromic O or chromic 1, non-boilable twenty day suture.

Suture Material Discussion. A detailed review regarding the action of various types of suture materials within the body will not be attempted here. It has long been known that small amounts of silk buried within tissue in non-infected areas cause relatively little reaction of the tissue. One of us has, for some time, used silk rather freely in doing herniorrhaphies and very fine silk for superficial ligatures in some thyroidectomies, feeling, as do some others, that less reaction occurs from silk in many tissues than from catgut. Experimental work which we have recently published elsewhere indicates, however, that some care must be used in the selection of silk, else adhesions and inflammatory reaction within the tissues may be more pronounced than with catgut. Some of the widely advertised specially treated silks should not be used routinely in the peritoneal cavity.[2]

Graves, the gynecologist, recommended the use of No. 7 braided silk doubled as suture in the Olshausen method of uterine suspension, believing that the knot could be tied more tightly than with catgut.[3] He remarked that heavy silk of this size might at times cause temporary sinus formation until discharged. One of us experienced this annoying but not serious complication previously on more than one occasion following the use of this material in the Olshausen operation and reverted to the use of catgut when this operation was done. It does seem that the use of unduly heavy strands of silk in tissues or the use of an undue quantity may be contraindicated.

COMMENT

Logic and past experiences would seem to confirm the experimental evidence

presented in Tables I and II. A fine strand of silk in the small quantities needed in invagination of the appendicial stump

material is definitely a more satisfactory material than catgut for use in invaginating the appendiceal stump. We also would call

FIG. 1. This shows clearly the similarity which is frequently found between the appendix in the dog and that in the human. In this animal the ligature was placed around both the meso-appendix and the appendix base. It was possible, in a number of other animals, to remove the entire appendix and bury the stump without any dissection whatever. (Photograph by DeGroat.)

FIG. 2. The clean end-result of the ligation and invagination by black silk purse-string suture as obtained in the specimen shown in Figure 1. (Photograph by DeGroat.)

would, theoretically, cause less reaction of the peritoneum than the catgut purse-string which is frequently used by surgeons. If this be true, it would certainly seem that there would be less likelihood of adhesion formation around the stump site and less likelihood of abscess or fistula formation if silk rather than catgut were used.

The literature regarding the history of catgut suture has recently been reviewed by Rhoads et al.[6] Different methods of preparation are used by different manufacturing firms. The popular chromic catgut suture is treated with chromic acid, iodine and sometimes other chemicals which harden it, causing it to be absorbed more slowly.

The comparisons in Table II speak for themselves. Not many animals were used in this series, but the experiments were carefully performed and readings were carefully and impartially made. We feel that the various factors mentioned in the brief review above, together with the results of these experiments would certainly indicate that a reasonably fine silk suture

attention to the fact that ordinary sewing silk was used in these experiments and would express the opinion that this silk is probably as good for purse-string suture as more expensive types of silk. We have had no experimental experiences with paraffinized silk but would consider it less desirable than plain sewing silk.*

It will be noted in Table I that where catgut was used there was a rather high percentage of animals in which the breaking down of the appendicial stump area occurred with subsequent peritonitis; whereas there was no instance where this complication occurred when silk was used. This breakdown occurred undoubtedly in this small series in a much higher percentage of instances than occurs in the human when catgut is used. It may be that this is due to the fact that burying the stump in the dog is sometimes more difficult than it is in the human. It is, therefore, occasionally necessary to use a slightly larger amount of catgut and slightly more tension than in the human. This factor, together with added inflammation caused by catgut, may have been

* Experiments which we have completed and published since this article was submitted for publication indicate that, as stated above, some of the specially treated expensive silks are definitely less suitable than cheaper grades of black silk.[2]

responsible for the high incidence of complications in the catgut group. The stump in the average dog, too, is broader and the ligature is a little more prone to slip off than in the human.

In fairness, a word must be said in interpreting the amount of adhesion formation found in the non-invagination group. The appendix of dogs is, as said, frequently broader and larger than in the human. The non-invaginated stumps in this experimentation were consequently frequently larger, sometimes leaving somewhat more mucous membrane exposed. We definitely do not believe, however, that this factor is sufficient to detract markedly from conclusions which have been made in comparing the three groups in the tables.

Correlation of the microscopic and gross evidence as related in our previous article,[1] would definitely indicate that there is some low grade inflammatory reaction in the buried stump region when even the silk purse-string is used. This is, however, insufficient to cause a breaking down of the area or a marked reaction of the overlying peritoneum. We believe that the absorptive power of the peritoneum itself in the small pocket about the stump is entirely sufficient to take care of a properly treated and properly buried stump. Previous studies of the laws of adhesion formation have definitely established that adhesions are almost certain to occur about any unperitonealized surface within the peritoneal cavity. True, frequently a meso-appendix may be manipulated in such a way as to cover completely or partially an unburied stump, but in the rather extensive clinical experience of one of us with the non-invagination technique, we would say that it is frequently true that the meso-appendix cannot be so manipulated. We have been reluctant to practice the tying of the meso-appendix in the same ligature used to ligate the appendiceal stump, as is sometimes recommended, for fear the tension sometimes created by this procedure might possibly predispose toward the slipping off of the stump ligature.

We have had no experience with the non-ligation and *inversion* method. In improved form it has prominent advocates

Fig. 3. The method we sometimes used in removing the free appendix tip as described under Method of Experimentation. If this appendix had been dissected free all the way to its base, raw surface would have been exposed which could have been covered only with difficulty. Bleeding, difficult to control without excess trauma and suture material, would have been encountered and future readings regarding adhesion formation as related to the objective dealt with in this article would have been unsatisfactory. The appendix lumen being large permitted us to remove the free tip which extended to about the area pointed to by arrow A. The stump was inverted into the lumen of the remaining part of the appendix (arrow B). The true base of the appendix in this instance, is situated at arrow C. Only a very thin band of adhesions (less than grade 1) was present at the buried stump region several weeks after the original operation was done (arrow C). (Photograph by Cowan.)

and for that reason should be given serious consideration.[5] Without further discussion of this technique, we will say that we, at present, favor the ligation purse-string method.

SUMMARY AND CONCLUSIONS

1. One of the authors, after having used the non-invagination or ligation and drop method of doing appendectomies for eight years, has returned to the use of the silk purse-string after having done the experiments analyzed here.

2. An analysis of experimental work upon three groups of animals in which the appendix stump was ligated and non-

TABLE I

CATGUT PURSE-STRING GROUP*

Date of Operation	Date Killed	Days	Gross Adhesions	Dog Number	Remarks
10-22-37	10-22-37	0	N.G.	85	Did not recover from anesthesia.
7-20-37	Died on table	0	N.G.	77	
6-21-37	6-22-37 Died	1 Died	N.G.	63	Slight contamination. Some erosion of purse-string suture.
6-22-37	6-24-37 Died	2 Died	Necrosis	66	Slight contamination. Necrosis and opening of lumen at operative site.
10-20-37	10-24-37 Died	3	4 Peritonitis	84	Local peritonitis considerable.
6-22-37	6-26-37 Died	4 Died	Peritonitis	65	Peritonitis necrosis, slipping of ligatures.
6-16-37	6-20-37 Died	4 Died	Peritonitis	59	Slight contamination; death with pus, local peritonitis, loosening of ligatures.
6-16-37	6-21-37 Died	5 Died	N.G.	60	Died. Some loosening of ligatures and local peritonitis.
7-16-37	7-21-37 Died	5 Died	Peritonitis	76	Slight contamination at operation. General peritonitis, appendix site opening.
7-13-37	7-19-37 Died	6 Died	3	73	Died. Eviscerated self; necrosis at site.
9-17-37	10-2-37	15	2½–3	81	1–2 drops pus at site.
6-22-37	7-8-37	16	2½	64	
6-21-37	7-8-37	17	3	62	
9-15-37	10-2-37	17	2½ and some miscellaneous	80	Small subserous hematoma at operation.
12-20-37	1-9-38 Died	20	2 plus	89	
12-13-37	1-12-38	30	1¾	88	
12-13-37	1-12-38	30	1	87	
9-22-37	10-21-37	30	2	82	Slightly more needle trauma than usual.
12-9-37	1-12-38	33	1 —	86	
7-20-37	8-25-37	35	0	78	
6-28-37	8-4-37	36	2½–3	68	
6-28-37	8-4-37	36	2	67	
7-16-37	8-25-37	39	0	75	
7-13-37	8-23-37	40	1	74	Drop of phenol on adj. ileum, neutralized with alcohol.
7-13-37	8-23-37	40	1	72	
6-15-37	7-28-37	43	2¾	58	Some difficulty peritonealizing. Considerable catgut.
6-16-37	8-4-37	48	1½–2	61	
7-1-37	8-23-37	52	2	71	Some more trauma than usual.
7-1-37	8-23-37	52	1¾–2	70	Slight contamination.
7-20-37	9-16-37	56	½	79	Slight contamination. 3–4 knots out.

* It is to be noted that we have not incorporated here a repetition of rather extensive tables which have appeared elsewhere.[1] The above table refers only to the complications which arose when catgut was used to bury the appendicial stump. In Table II a summary compares the degree of adhesion formation which occurred in the above group with the adhesions which occurred in the silk purse-string group and the non-invagination group. Complete tables regarding the latter have appeared in detail previously.

invaginated, invaginated with silk purse-string sutures and invaginated with catgut purse string sutures is presented.

3. Adhesion formation seemed to be less frequent and extensive where the stumps were invaginated with "untreated" black

TABLE II

	Silk Purse-String	Catgut Purse-String	Not Invaginated
No adhesions...........	7	2	0
Less grade 1............	15	4	2
Grade 1 to 2 inclusive....	6	9	2
Grade 2 or over.........	5	11	17
Total adhesion readings..	26	22	26

It may be noted that in the catgut purse-string group only twenty-two adhesion readings are listed. We feel that twenty-six good experiments were made, however. Referring to Table 1, it may be seen that in some of the animals peritonitis developed in association with a loosening of the sutures and ligatures about the buried stump. In four of these animals we felt the technique was not at fault and that the openings of the stumps giving the general peritonitis were of significance. (See Comments.) Slipping of ligatures occurred in a lesser number of instances in the non-invagination group but in these instances short stumps and technique, we feel, may have been responsible.

silk purse-strings; most frequent when the stumps were not invaginated; and intermediately frequent when catgut purse-strings were used.

4. Other complications were more frequent in the group in which catgut was utilized but for reasons discussed, one should be guarded in interpretation of this · finding. The possibility that the catgut purse-string may, in the human, be more likely to predispose to a higher incidence of abscess and fistula formation than occurs with the non-invagination technique, is to be considered; but we feel that it would take further investigation to settle this particular point.

5. A brief review citing the literature and experiences of others indicating the irritating nature of hardened catgut is given.

6. The non-irritating qualities of fine silk in small quantities, as experienced in the clinical and experimental work of the authors, as well as in the hands of others is mentioned.

7. We believe that the microscopic evidence reviewed in a previous presentation,[1] correlated with gross experimental findings in experiments in which the stump is invaginated with the silk purse-string suture indicates that the absorptive power of the peritoneum in the small pocket about the buried stump is sufficient to take care of an appendicial stump which has been properly ligated, treated, and invaginated with the proper type of black silk, without predisposition to abscess formation or increased adhesion formation. That this is true is indicated not only by the experiments analyzed, but also by our own past experimental and clinical studies in adhesion formation, as well as by studies of the characteristics of the peritoneum by others.

REFERENCES

1. Donaldson, J. K., and Thatcher, H. S. Clinical and experimental studies with non-invagination of the appendicial stump. *South. M. J.*, 31: 488 (May) 1938.
2. Donaldson, J. K., and Cameron, R. R. A study of the use of silk, catgut, and the Noble plication with reference to abdominal adhesions. *Surgery*, 5: 511 (April) 1939.
3. Graves, W. P. Gynecology, 3rd ed. Philadelphia, 1924. W. B. Saunders Company.
4. Longacre, J. J. Experimental total pneumonectomy. *J. Thor. Surg.*, 4: 587–606 (Aug.) 1935.
5. Ochsner, A., and Lilly, G. The technique of appendectomy. *Surgery*, 2: 4: 522 (Oct.) 1937.
6. Rhoads, J. E., Hottenstein, H. F., and Hudson, I. F. The decline in the strength of catgut after exposure to living tissues. *Arch. Surg.*, 34: 377 (March) 1937.
7. Shambaugh, P., and Dunphy, J. E. Postoperative wound infections and the use of silks; an experimental study. *Surgery*, 1: 379 (March) 1937.

FRACTURE DISLOCATION OF THE ELBOW WITH DISPLACEMENT OF THE INTERNAL EPICONDYLE INTO THE JOINT

ADOLPH A. SCHMIER, M.D.

Adjunct Orthopedic Surgeon, Hospital for Joint Diseases; Assistant Adjunct Orthopedic Surgeon, Sydenham Hospital

NEW YORK CITY

DISLOCATION of the elbow with displacement of the internal epicondyle into the joint is not commonly seen. In those cases reported with nerve disturbance, it has always been the ulnar nerve involved, while in the case here described the median nerve was affected. Employing the closed method of reduction similar to the procedure previously reported by me, the result obtained was very favorable from the anatomic, neurologic and functional viewpoints.

M. S., a boy 16 years old, was playing basketball on December 21, 1937. He fell backward on his right elbow and was immediately incapacitated. He was seen within two hours of his injury and the elbow was markedly swollen but only slightly painful. The forearm was displaced considerably laterally and posteriorly. The relation of the olecranon and humeral condyles was disturbed, the olecranon being displaced posteriorly and medially. The lower end of the humerus was prominent anteriorly and filled the cubital fossa. The forearm was markedly abducted at the elbow and shifted laterally in a translatory fashion. The radial pulse was good and motion of the thumb and fingers was normal. Sensory examination revealed slight hypesthesia of the volar surfaces of the distal phalanges of the thumb, index and middle fingers.

Roentgenograms of the right elbow (Fig. 1) disclosed a posterior, lateral and upward dislocation of the radius and ulna en masse; also a separation of the epitrochlear epiphysis with its displacement into the elbow joint. Abduction of the forearm could also be noted.

Under general anesthesia the method of closed reduction described in a previous report was employed. Briefly, the method is based on the fact that the superficial flexors and pronator of the forearm originate by a common tendon from the epitrochlea. If tension is applied to these muscles by supinating the forearm, extending the elbow, wrist and fingers, and slightly abducting the forearm in order to increase the gap between the trochlea and the ulna, the epitrochlea might be dislodged from the elbow joint by the muscle pull upon it. This case differed from the one previously reported, however, in that the forearm was displaced upward instead of downward. Because of this, complete extension at the elbow could not be obtained. In addition to the other maneuvers, considerable traction was therefore employed until the coronoid process of the ulna passed the lower end of the humerus. Complete extension at the elbow was then possible and the epitrochlea was maneuvered out of the joint. However, the lateral deformity at the elbow remained. The translatory lateral shift of the forearm was then corrected by gentle pressure directed on the internal humeral condyle laterally, and pressure on the radius and ulna toward the medial side. The elbow then clinically assumed its normal appearance. A posterior plaster of Paris splint was applied from the axilla to the hand with the elbow at right angles. The radial pulse remained good.

Roentgenograms (Figs. 2 and 3) taken the following day revealed the epicondyle in its
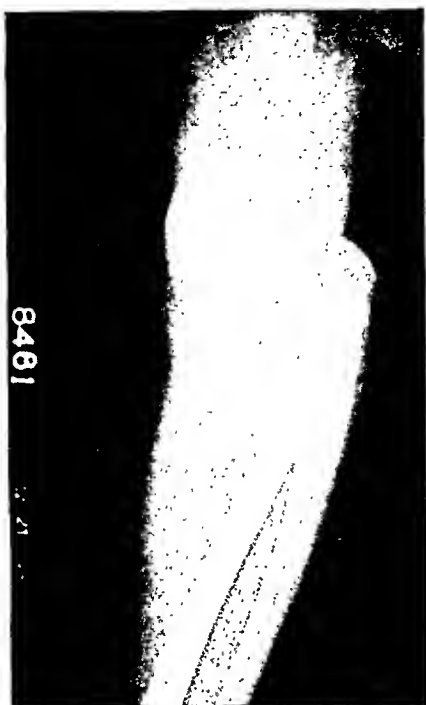
Fig. 1. Anteroposterior and lateral roentgenograms showing posterior, lateral and upward dislocation at elbow with intra-articular interposition of epitrochlea.
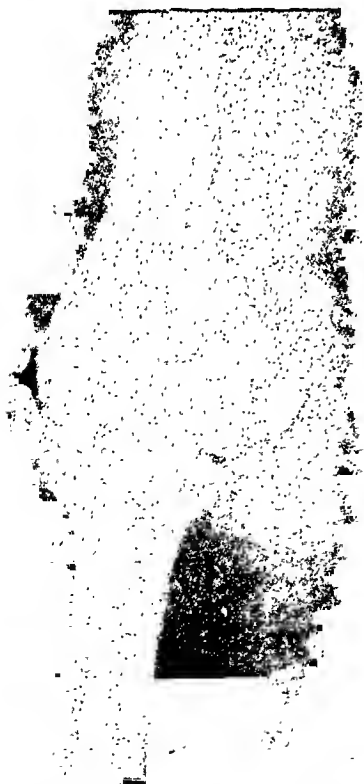
Fig. 2. Anteroposterior view following reduction. Epitrochlea in normal position.

Fig. 3. Lateral view following reduction.

normal position and the elbow completely reduced. The radial pulse was still good and there was no cyanosis or swelling of the hand.

FIG. 4. Four weeks after reduction. Evidence of some osseous proliferation between epicondyle and humerus. Note periosteal reaction along the external condyle.

The patient had no discomfort at the elbow and his only complaint was a tingling of the volar surfaces of the distal phalanges of his thumb, index and middle fingers. Sensation to touch was diminished. Motor power was good. He was discharged from the hospital on December 24, at which time the hypesthesia was diminished.

On January 3, he was again seen in the dispensary. The median nerve sensory disturbance had entirely disappeared. Motion of the fingers and thumb was normal and he had no complaints referable to his elbow.

Twenty-four days following reduction, the posterior plaster of Paris splint was removed. Some periarticular swelling was still present at the elbow, but there was no tenderness. The internal epicondyle could be felt firmly attached and there was no false motion or crepitus.

Motion at the elbow was immediately possible as follows: AGE = 120 degrees; AGF = 75 degrees. Short wave diathermy, massage and exercises were instituted.

On January 19, active motion was present from 75 degrees of flexion to 130 degrees of extension.

On January 28, swelling at the elbow was gone. Flexion at the elbow was 10 degrees short of normal and extension was possible to 155 degrees. Pronation and supination were normal. There was no pain or tenderness at the elbow and sensation in the hand was normal.

On February 2 a roentgenogram (Fig. 4) revealed the internal epicondyle in normal position, with some evidence of bony union with the humeral condyle. There was also noted a periosteal proliferation along the external humeral condyle. This was probably due to injury of the periosteum either at the time of accident or during the reduction. This finding was also noted in the case previously reported, but it had no effect on the motion at the elbow almost four years later. Similar periosteal reaction is described in cases treated by open reduction.

By February 16, flexion, extension, pronation and supination at the elbow were normal. The patient had no complaints and there were no sensory disturbances—he was using his right arm painlessly.

On April 1, the patient stated that he was able to play handball and baseball without any difficulty. Clinically, the contour of the elbow was normal. Motion of the right elbow equalled that of the left, as follows: AGF = 40 degrees; AGE = 180 degrees; pronation was 75 degrees and supination 95 degrees.

Following the publication of my first case, I received a communication from Mr. Norman Roberts of Liverpool calling my attention to his report of four cases reduced by the closed method. Since his report he has treated four more cases in the same manner. Some of his cases presented ulnar nerve involvement, but there was none with median nerve disturbance.

I am greatly indebted to Dr. Harry Sonnenschein, on whose service the patient was treated, for his kind permission to report the case.

REFERENCES

COTTON, F. J. Dislocations and Joint-Fractures, 2nd Ed. Philadelphia, 1924. W. B. Saunders.

Cotton, F. J. Elbow dislocation and ulnar nerve injury. *J. Bone & Joint Surg.*, 11: 348 (April) 1929.

Dunlop, J. Traumatic separation of the medial epicondyle of the humerus in adolescence. *J. Bone & Joint Surg.*, 17: 577 (July) 1935.

Fevre, Marcel, and Roudaitis. La réduction non sanglante des fractures de l'épitrochlée avec interposition de ce fragment dans l'interligne articulaire du coude. *Rev. d'orthop.*, 20: 300, 1933

Roberts, N. W. Displacement of the internal epicondyle into the elbow joint. Four cases successfully treated by manipulation. *Lancet*, 78 (July 14) 1934.

Roberts, N. W. Personal communication.

Schmier, A. A. Fracture dislocation of the elbow with ulnar nerve involvement. Reduction by tension on the superficial flexor group of muscles. *J. Bone & Joint Surg.*, 18: 1030–1035 (Oct.) 1936.

Zadek, I. Fracture of medial epicondyle of the humerus with its displacement into the sigmoid cavity of the ulna. Report of two cases. *Am. J. Surg.*, 39: 217, 1925.

There are probably more fractures of the upper than of the lower extremity. Fractures of the phalanges and ribs are probably most numerous. Following these, without any attempt to place them in order of frequency, the common fractures are of the lower extremity, the radius, the region of the ankle, the metacarpals, the shaft of the bones of the leg, the skull, clavicle and humerus.

From—"A Textbook of Surgery," edited by Frederick Christopher (Saunders).

INJURY TO THE LONGITUDINAL SINUS ACCOMPANYING A DEPRESSED FRACTURE OF THE SKULL

GASTON A. CARLUCCI, M.D., F.A.C.S.

Associate Attending Surgeon, Bellevue Hospital; Attending Surgeon, Misericordia Hospital

NEW YORK CITY

INJURIES to the longitudinal sinus are seldom encountered, and the few that are seen are always fatal.

Since recovery took place in the following, it seemed sufficiently interesting to report in detail.

S. H., 32 years old, a male garage attendant was struck by a truck on November 9, 1937, while riding on a motorcycle delivery wagon. He was knocked unconscious and brought to the Misericordia Hospital shortly after the accident occurred at 7 A.M.

He slowly regained consciousness, but remained hazy about his accident.

There were a hematoma of the scalp, some abrasions of the shoulders and a contused wound of the right heel. The pupils were widely dilated. There was definite weakness of the right upper extremity and a positive Babinski on the right side. All other reflexes were unchanged.

At 2 P.M. of the same day, the patient was stuporous and suddenly had an attack of projectile vomiting, bringing up about 20 ounces of greenish fluid. The blood pressure throughout the day remained about the same, 130/70. Spinal tap showed fluid under moderately increased pressure, but clear.

The following day, the hematoma of the scalp was increasing in size; the patient was conscious but complained of a severe headache. Motion of the right forearm was distinctly weak and the right grip was nearly all gone. Other reflexes were normal except for the persistent right Babinski.

An x-ray by Dr. R. Pound on the day of accident showed a fracture of the vertex of the skull in the region of the coronal suture. The right parietal bone seemed to be elevated on the distal end and the frontal depressed. The posterior portion of the sagittal suture was separated and the anterior margin of the right parietal bone overrode the posterior margin of the frontal.

In view of the x-ray findings and the beginning loss of power of the right upper extremity, etc., it was felt that a decompression was indicated. This was done under gas-oxygen anesthesia, the patient lying on his back with the head slightly elevated. A 3 inch longitudinal incision was made over the vault of the skull, the aponeurosis markedly infiltrated with blood was split and the extensive т fracture exposed. The frontal fragment was well driven under the posterior one and through the line of fracture there was a steady oozing of blood. A trephine opening, about ¾ inch in diameter was made in the center of the overlying bone and this allowed the introduction of a periosteal elevator to act as a lever to realign the fracture. This was done, but as a result, active bleeding began from the bone edge and from a small tear on the dorsal surface of the longitudinal sinus immediately underneath the anterior fragment. Bone wax easily stopped the bleeding from the bone but a small trephine opening had to be made in the anterior fragment to expose the sinus tear better. This evidently released what bony pressure there was on the sinus and blood began to pour out profusely. Several attempts were made to control the bleeding with packing, but to no avail. It was then decided to suture and tie the longitudinal sinus transversely, anteriorly and posteriorly to the laceration with two silk sutures. This was done, keeping pressure on the opening as much as possible, and the bleeding stopped.

Actually, however, as the attempt was made to control the hemorrhage, the shock was so great that the pulse became imperceptible and the blood pressure could not be gotten. Heroic stimulation had to be given with 50 per cent glucose solution intravenously, etc. The wound was then packed quickly with sterile gauze and most of it closed. A transfusion of 500 c.c. of whole blood followed. The patient gradually rallied and was finally sent back to the ward.

The next day the patient remained stuporous and could only move his lower extremities

slightly. On the second day, his temperature rose to 103; he became restless and his extremities, particularly the lower ones, became

FIG. 1. Lateral x-ray, showing depressed fracture of vault of skull.

spastic. It was thought, therefore, that this was the result of increasing intracranial pressure as a result of the tight packing. The wound was dressed and some of the packing was gently removed. Shortly after, the restlessness subsided and the temperature dropped.

Over a period of five days the rest of the packing was removed without any bleeding and the patient slowly but steadily became rational. The spasticity of the extremities disappeared so that on the twenty-second day after the accident he was able to get up in a chair. The wound has since healed well, except that one of the silk sutures gradually worked itself out. Twelve weeks after the accident the patient was walking about, with mentality clear, and the only residual symptom vertigo on arising or suddenly stooping. The wound healed, but a definite defect could be felt in the vault of the skull where cerebral pulsation was transmitted.

X-rays taken on November 18 were reported as showing a circular area of bone removed from the vertex of the skull just posterior to the coronal suture. "The previous deformity has been eradicated."

The interesting features of this case are:

1. The extensiveness of the fracture and depression with recovery to consciousness

FIG. 2. Anteroposterior view, showing fracture and separation of coronal suture.

after the accident and the slow development of paralysis of the upper extremity. This was probably due to the fact that the extravasating blood had found a passage into the scalp aponeurosis and was, therefore, causing very little increase in the intracranial pressure.

2. The active bleeding of the sinus as soon as the pressure is released on it.

3. Although the hemorrhage was not really exsanguinating, the blood pressure dropped and the patient very quickly went into shock.

4. The development of signs of spastic paralysis after the operation which cleared up on the removal of the packing.

5. The wound and the dura healed and the patient regained his mental faculties.

As far as can be determined, a fracture of the vault of the skull accompanied by a laceration of the longitudinal sinus is not common. When it does occur, the injury is so severe that the patient, as a rule, does not survive. Very few patients who have recovered are reported in the literature.

In the American periodicals, only Bagley[1] gives a very minute report of two cases, one of which survived. This man,

aged 21, had a small depressed fracture of the external occipital protruberance. In his other case no fracture could be

lesion of this sinus. He also quotes Ronge-mont and Robin, who describe the autopsy findings in their case.

FIG. 3. Lateral view, showing depression corrected.

FIG. 4. Anteroposterior view, showing trephine opening over longitudinal sinus.

FIG. 5. Elevation of anterior frontal fragment.   Close examination shows the line of fracture.

determined by x-ray, but at autopsy a thrombosis of the sinus was found.

Chavanne[2] reports that in 1930 Gau collected sixty-seven cases of traumatic

Doench[3] reports an unusual case of death following air embolism. Under normal conditions, he states, air embolism of an injured sinus is not possible because

there is a positive pressure of at least 90 mm. Hg when the patient is in the horizontal position. However, conditions

compress was then momentarily removed and at this moment the characteristic crackling sound of air embolism was heard

Fig. 6. Flow of blood from longitudinal sinus and attempt to suture the tear.

are quite different when dealing with an exsanguinated patient in whom the pressure conditions are changed. If the patient is not in the horizontal position, but is operated upon in the sitting position, the pressure falls. The case reported by him was that of a child, 11 years old, with a wound 10 cm. long in the occipital region running obliquely over the middle of the skull. X-ray revealed a large depressed fracture. The child was conscious but presented the appearance of having lost much blood. Under ether anesthesia, the wound margins were excised and the depressed fracture exposed. Removal of the largest bone fragment caused a severe hemorrhage from a 3 cm. tear in the longitudinal sinus. Immediate tamponade with gauze was done, as, owing to the infected wound and the badly crushed dura, suture was out of the question. As several other splinters of bone, turning inward, had to be removed the author decided to make a double ligature. During the operation the pulse had become markedly weaker. The anterior suture was done first. To introduce the posterior suture the trephine incision had to be extended. The

and air bubbles formed at the site of the sinus injury. Immediate collapse followed, and in spite of all attempts to save him, the boy died.

Doench is of the opinion that the preceding hemorrhage and the raised position of the child were, no doubt, contributing factors in the causation of air embolism. The chief cause, however, lay in placing the ligature first in the frontal region. In the state of negative pressure thus engendered by the position and hemorrhage of the patient, air instead of blood was sucked in.

Gucci[4] brings out several interesting facts. Hemorrhage is the great complication of dural sinus wounds, being usually profuse and rapidly fatal unless checked. The sinus walls have no smooth muscle fiber and few elastic elements; there is also close adherence to the bone. In sinus lesions one may observe that (1) notwithstanding laceration, the blood does not escape. This is due to the fact that the adherence to the bone wall is sometimes so strong that the blood cannot loosen the dura mater and in itself acts as a tampon. (2) The blood may flow and turn to rapid fatal hemorrhage. Only quick aid can

prevent death. (3) The blood may infiltrate above or below the dura mater. (4) The blood loosening the soft aponeurosis forms a subcutaneous hematoma remaining in connection with the sinus. (This apparently had happened in my case.) He also states that in cleansing the wound or repairing the tear, care should be taken to prevent death from an air embolus.

All the writers agree that digital pressure should be used as a provisional measure to combat hemorrhage. To control it, tamponing is considered efficacious, but suture of the sinus is advised if possible, when the tear is small and the wound clean. Suture may not be rapid enough, but if it can be used provides not only hemostasis but recanalization of the sinus. When tamponing only is used, extreme care is necessary on removal in order to prevent secondary hemorrhage.

SUMMARY

A case of trauma to the longitudinal sinus following a depressed fracture of the vault of the skull with recovery is reported.

The salient facts are: the slowness of the onset of symptoms of increasing intracranial pressure probably due to the fact that the extravasated blood leaked out into the scalp aponeurosis; the development of acute shock as soon as brisk bleeding occurred from the torn sinus when the depressed fracture was lifted up into place; the importance of being prepared to give heroic stimulation when this shock occurs; the necessity to reduce the packing as soon as symptoms of increasing intracranial pressure develop, as evidence by the increasing spastic paralysis of the extremities; and, finally, the remarkable recovery with very few residual symptoms after such an extensive injury.

REFERENCES

1. BAGLEY, L. C., JR. Traumatic longitudinal sinus lesions. *Surg., Gynec. & Obst.*, 58: 428–502 (Feb. 24) 1934.
2. CHAVANNE, M. F. Hémorragie du sinus longitudinal supérieur. *Lyon-méd.*, 150: 545–547, 1932.
3. DOENCH, J. O. Luftembolie bei Verletzung des Sinus longitudinalis. *Zentralbl. f. Chir.*, 60: 486, 1933.
4. GUCCI, G. Therapy of wounds of the superior longitudinal sinus. Case. *Policlinico (sez. prat.)*, 41: 414–421, 1934.
5. CECCHINI, N. Traumatic rupture of superior longitudinal sinus: lateral suture in case. *Gazz. internaz. med. chir.*, 46: 126–128, 1936.

OSTEOGENIC SARCOMA OF THE ASTRAGALUS

REPORT OF A CASE

S. Ralph Terhune, M.D., F.A.C.S.

Attending Orthopedic Surgeon, Hillman and Birmingham Baptist Hospitals

BIRMINGHAM, ALABAMA

THE rarity of osteogenic sarcoma in the small bones makes the following case worthy of interest.

Mrs. A. R. (patient of Dr. C. D. Gaines), white, 34 years of age, was first seen by me September 17, 1937.

In February 1937, the patient had fallen from a porch about four feet high, injuring her left ankle. Pain in the ankle remained constant and by July prevented weight bearing.

Examination revealed enlargement of the foot and ankle with very little motion in the ankle. There was a small hard mass over the dorsal surface of the astragalus which was markedly tender. There was some atrophy of the left thigh and calf. A roentgenogram showed a tumor mass projecting from the upper surface of the neck of the astragalus with destruction of the cortex. Chest plates were negative. The Wassermann test and the urine were negative.

Blood count showed: red blood cells—4,200,-000; hemoglobin—75 per cent; white blood cells—8,100. Polymorphonuclear neutrophiles were 62 per cent; large lymphocytes, 4 per cent; and small lymphocytes, 34 per cent.

On September 18, a biopsy was done under ether anesthesia. The pathologist, Dr. G. S. Graham, reported the tumor to be a low grade osteogenic sarcoma.

On September 29, 1937 an amputation at the junction of the distal and middle thirds of the thigh was performed.

The pathologic report by Dr. Graham was as follows: "The specimen consists of a left leg amputated through the lower third of the femur. There is a recent healing surgical wound of the upper lateral dorsum of the foot just anterior to the internal condyle. The foot and tibia were sectioned longitudinally. The wound mentioned extends inward to the surface of the astragalus. Upon the bone surface here, there is an elevated mass of roughened bone, and bone formation is present in the soft tissue of

the joint wall. The body of the astragalus shows an irregular, dull, red discoloration and vaguely defined areas of sclerosis. Upon the sides of the

Fig. 1. Longitudinal section of foot and ankle.

bone opposite the wound there is a larger mass of rough bone which projects across the joint cavity into the soft tissue at the anterior inferior angle of the tibial joint surface. An irregular fibrillar tissue extends downward over the antero-inferior surface of the astragalus. Here, the cortical layer of the bone is destroyed over its entire antero-inferior surface.

"Adjoining the rough bone mass mentioned above, there is a nodulated tumor mass 2.5 cm. long and 1.5 cm. in diameter that lies in the soft tissue over the tibio-astragalar joint. It is in part milk white, soft, homogeneous, and of typical fish-flesh appearance. Closer to the bone, however, it is slightly yellowish and con-
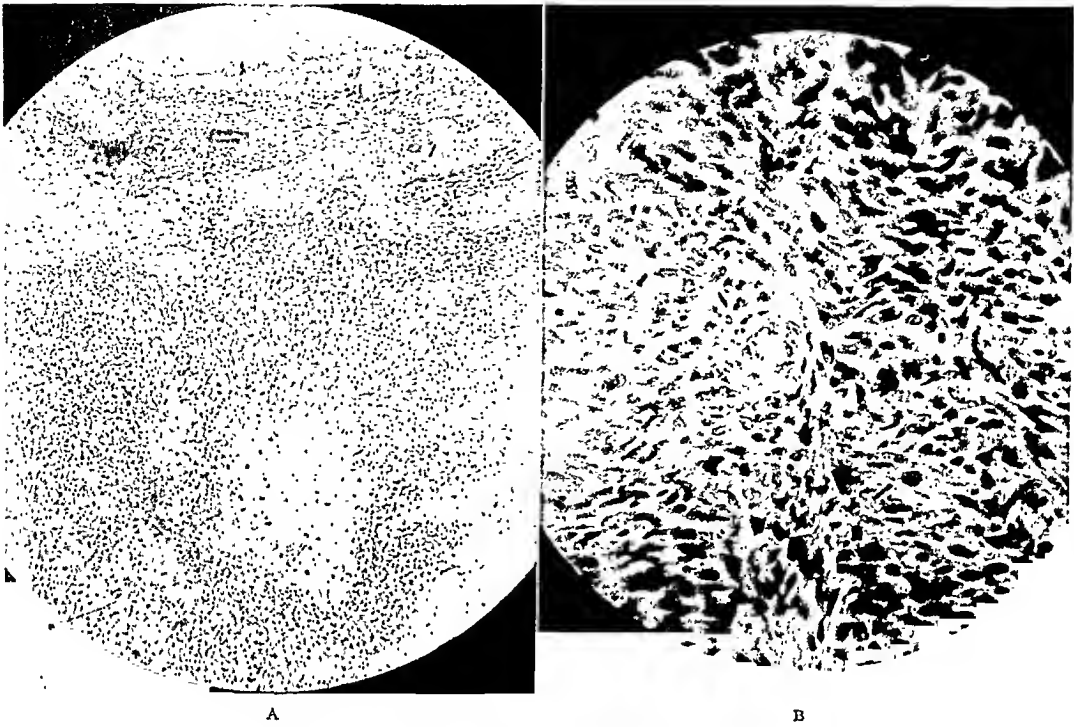
FIG. 2. A, low power microscopic section. B, high power.

FIG. 3. Lateral view of sarcoma.

tains fine bone spicules. Below this segregated nodule another tumor mass in the soft tissue extends downward for a distance of 3.5 cm. in width and 2 cm. in depth. Here, the tissue is dark red to dull yellow, firmer, somewhat translucent and contains numerous bone spicules. Longitudinal section of the leg bones and the femur shows no evidence of tumor invasion.

"Microscopic examination of sections of the astragalus shows in part of its extent a spongiosa of natural appearance. In a considerable part of the block the marrow spaces are filled by a tumor tissue usually consisting of fibroblasts. These are arranged in closely crowded groups and columns and are large with hyperchromatic nuclei and occasional giant nuclear forms. Mitotic figures are present but not particularly frequent. In some fields there are small patches of atypical cartilage and occasionally new formed bone within the cartilage. A large area at one side of the block shows necrosis of bone and infiltrating tumor. Section of the tumor mass described in the soft tissue outside the bone shows in the soft portion a highly cellular fibroblastic structure with occasional traces of new formed bone. In the firmer portion the mass contains a large admixture of cartilage and more frequent bone trabeculae. This area also shows large numbers of mitotic figures.

"Pathologic diagnosis: osteochondrofibrosarcoma of the astragalus."

The wound promptly healed and the patient was discharged from the hospital on October 12, 1937. Follow-up examinations to May, 1939 show the patient in good condition with no complaints.

Preceding this case, the Registry of Bone Sarcoma of the American College of Surgeons had only four similar cases. The history in each was practically the same as in this one in that there was a lengthy interval from onset until the time of diagnosis. The histology in this instance as in the others would seem to indicate a better prognosis for life than is usual in osteogenic sarcoma of the long bones.

MALIGNANT PAROTID TUMOR IN THE NEWBORN

H. A. McKnight, M.D., F.A.C.S.

Surgeon, Springfield City Hospital

SPRINGFIELD, OHIO

PAROTID tumors may occur at any age and cases have been recorded in the literature at ages from 8 to 80.

Fig. 1. Following operation, quick return of tumor. Shows mass growing in auditory canal.

Pailler reported a tumor in a girl of 11 months; McFarland, one in a child less than 1 year of age, and Wood, a rapidly growing tumor in a child of 7 months. These reported tumors occurring in children were the so-called mixed tumors of the parotid gland, but we can find no records of carcinoma of the parotid in very young children. In the case we have to report, the mother insists the swelling below the ear was present at birth.

The patient, aged 15 months, a well nourished black infant, was first admitted to the Springfield City Hospital June, 1935. A freely movable, hard, encapsulated mass about the size of an olive, irregular in shape was observed behind and below the left ear. It was not tender nor fluctuant. The cervical glands on the opposite side of the neck were slightly enlarged, the ear drums normal.

Cultures of the throat showed staphylococcus and Streptococcus hemolyticus. Blood and urine examinations were negative. The temperature ranged from 97.3 to 101 degrees.

The surgeon who saw the child at this time, diagnosed the condition as branchial cyst, and because of the throat infection, sent her home.

In September of the same year, she was readmitted. The tumor mass was still present and had enlarged slightly, growing more rapidly during the last two months. The color of the skin over the tumor had changed to a reddish purple. The tumor was now about the size of a duck egg, still freely movable and nowhere attached to the skin or underlying tissues. It was, however, hard and indurated.

At operation a tumor mass was found posterior to the left ear, attached to the left parotid gland. It was very friable and grossly appeared malignant. The tumor and the lower part of the parotid gland were excised.

Pathologic Report. The tumor was the size of a plum, elastic and of medium consistency, grayish-white in color. It was covered by skin in an area 4 by 2 cm. On cut surface it presented an almost homogenous structure and a mucus-like fluid could be removed.

Another mass was of firmer consistency, of reddish-gray color and seemed to be surrounded with connective tissue. This proved to be normal parotid tissue.

Microscopic examination showed that a large amount of stroma had been developed—it was rather loose but rich in spindle-shaped cells very similar in form and grouping to myxomatous cells. In this stroma large strands of epithelial cells were scattered, frequently in a definite gland-like grouping similar to that of the ducts of the parotid and salivary glands. In many places the epithelial cells were grouped in rows, resulting in solid strands of tissue. The lining cells were very irregular in size and shape, some cells being extremely large. Mitotic figures and pyknotic nuclei were common. The diagnosis given was adenocarcinoma of the parotid gland. The child was admitted for the third time in December of the same year, with an admission diagnosis of tumor behind the left ear and hemorrhage from the left auditory meatus. On the day prior to admission the

patient had begun to bleed from the left ear, and this bleeding had since increased in amount.

temporal muscle and an extremely edematous brain were also noted. The left temporal lobe of the brain was entirely replaced by tumor
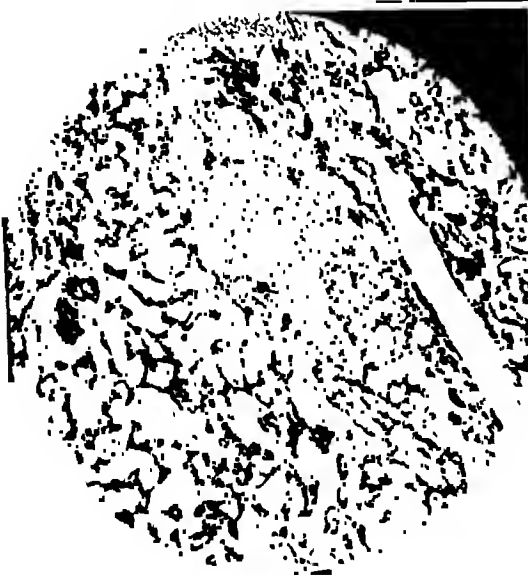
FIG. 2.

FIG. 3.

There was a decided droop of the left side of the face with paralysis of the facial muscles and dribbling of food and saliva from the left corner of the mouth. The external auditory canal was filled with a polypoid fungus mass which bled easily when touched. A mass the size of a lemon lay behind and below the left ear. It was hard and irregular in outline and only slightly movable.

A series of x-ray treatments was started and the tumor, while first increasing in size, later became smaller and then increased rapidly. Three months later the left eye developed a diffuse keratitis and a corneal ulcer, and a bloody discharge issued from the left nostril.

Blood was withdrawn for irradiation and reinjection into the veins. This was followed by a marked temperature reaction. Irradiated blood was reinjected several times with some improvement in the child's condition, but convulsions of the head and upper extremities with projectile vomiting developed and in April the child died suddenly.

Autopsy showed a male colored body 85 cm. long with extreme rigor mortis. There was marked edema of the left side of the face, with the left eyeball enormously protruded and the external ear swollen. Under the left ear was a scar 4 cm. long, and in front of the ear another scar 3 cm. long. Marked edema of the left

masses. Between the fossa cerebri media and the fossa cerebri anterior the bone had been entirely broken down by invasion of a tumor mass the size of a chestnut showing many hemorrhages. The os petrosum of the temporal bone was, in its anterior half, entirely destroyed by invading tumor masses. In the same way the sella turcica was entirely destroyed and replaced by neoplastic growth. Parts of the sphenoid which separated the fossa cerebri media and the fossa cerebri anterior were broken down and invaded by tumor and many hemorrhages. The left orbita was entirely filled with tumor mass.

Microscopic examination of the temporal lobe showed diffuse invasion and replacement by gland-like structures similar to the ducts of of the parotid gland. The cells were irregular in size and shape, and mitotic figures and pyknotic nuclei were common.

Examination of the tumor masses of the orbita showed extensive hemorrhages and the same structure as found in the temporal lobe. The gland-like structure was not always preserved, but the carcinoma diffusely invaded the stroma, forming solid strands of cancer tissue. Frequently thrombosis had occurred with tumor cells in the thrombotic vessels. The petrosal bone was invaded by strands of cancer cells like those in the brain.

The lungs showed multiple hemorrhages in the pleura and parenchyma, and several metastases the size of a lentil, yellowish and

FIG. 4.

dark red in color. Microscopic examination disclosed extended hemorrhages and lobular pneumonia with infiltration by polymorphonuclear leucocytes. In the areas corresponding to the metastatic nodules, the lung tissue had been replaced by the same strands of tumor cells in the gland-like groupings found in the brain. No metastases were found in the liver, kidneys or pancreas.

Final diagnosis was adenocarcinoma of the parotid gland, invading the petrosal, the sphenoid and sella turcica, with complete substitution of the temporal lobe by carcinoma and metastases in both lungs.

This case is of interest because of the early incidence of cancer. Although there is a fairly wide age distribution, the majority of these tumors appear in persons between the ages of 20 and 45 years.

Enlargement of the tumor is generally rapid. The mass is adherent to the skin or the underlying tissues. Although the differentation of benign from malignant tumors of the parotid gland is often extremely difficult the distinction can usually be made if the following points are kept in mind.

1. Duration of benign tumors is twice that of malignant tumors; the former about eight years, the latter four years.

2. Pain is more severe and more radiating in malignant tumors.

3. Benign tumors are encapsulated, rub-

FIG. 5.

bery and nodular growths without enlargement of the regional lymph nodes. The malignant growth is a more diffuse swelling adherent to the skin or underlying structures. It is either harder or softer than a benign lesion and there is associated enlargement of the lymph nodes.

Malignant epithelial tumors of the salivary glands are by no means rare. The carcinomata develop rapidly, and while at first they may be encapsulated, they soon invade the whole gland, the capsule and the regional lymph nodes. Distant metastases are rare.

Local recurrence after removal of parotid cancer is usually very prompt, a matter of months rather than years. Secondary operations have been uncommon, and according to Benedict and Meigs, only five patients have been operated on more than once.

In this case the extension to the skull was an extrusion of the primary growth through the fissure of Santorini into the auditory canal, then into the facial canal and the petrous portion of the temporal bone, to the brain. The secondary growths in the lungs were true metastases.

TOXIC NEURONITIS OF PREGNANCY

REPORT OF A CASE

Samuel Lubin, m.d., f.a.c.s.   and   Nathan W. Newman, m.d.

Attending Obstetrician, Cumberland Hospital   Assistant Attending Physician, Cumberland Hospital

BROOKLYN, NEW YORK

UP to within recent years toxic neuronitis was mentioned infrequently as a complication of pregnancy. Only since the report of Berkwitz and Lufkin[1] has the condition been more readily recognized. In addition, at about the time their report was presented, various other investigators, notably Strauss and McDonald,[2] associated toxic neuronitis with a vitamin B_1 deficiency. It was no longer considered solely a toxic disturbance, superimposed upon pernicious vomiting of pregnancy, but rather as a clinical entity dependent upon a specific avitaminosis. In fact, vomiting need not necessarily precede this complication of pregnancy, as was brought out by the report of Maisel and Woltman.[3] However, in the case which is presented here toxic neuronitis did follow pernicious vomiting of pregnancy, and this was also true of a case previously reported by one of the authors of this report (Lubin[4]).

The mortality associated with toxic neuronitis complicating pregnancy varies from 25 per cent, according to Berkwitz and Lufkin,[1] to 68 per cent, as shown by the figures compiled by Plass and Mengert.[5] However, most of the cases utilized to obtain these figures, occurred previous to the time that the etiologic relationship between vitamin deficiency and toxic neuronitis had been established. With this important advance in the knowledge of the condition, the future should be expected to disclose a definite reduction in the incidence mortality of this complication of pregnancy.

The method of treatment previously advocated had been termination of pregnancy. This shifted, however, and treatment has been directed more toward the use of vitamin B_1, especially since the latter has become so readily available and is so uniformly standardized. The swing in this direction seems warranted because of the excellent results attendant upon its use. Thus, cases have been reported by Vandel,[6] Theobald,[7] Fouts, Gustavson and Zerfas,[8] and Cook,[9] where the symptoms of toxic neuronitis have been relieved and the pregnancy carried successfully to term, by the sole use of vitamin therapy. If one carefully analyzes their reports, however, it will be noted that the majority of the cases were of a mild type. On the other hand, the patients reported by Stroh[10] and Bingham[11] did not react satisfactorily to a trial of vitamin B_1, with the result that therapeutic abortion had to be done. From the description, the cases of the latter authors were apparently further advanced than those of the former, which responded to vitamin therapy alone. In addition, we note the case of Forman,[12] the symptoms of which appeared following therapeutic abortion for pernicious vomiting of pregnancy. Improvement then resulted from the use of vitamin B_1 therapy.

It might be assumed that the use of either therapeutic abortion or a vitamin regimen, individually, does not solve the problem of the treatment of toxic neuronitis of pregnancy. However, the latter does offer an additional mode of attack and may, at least in early cases, prove to be the curative agent per se. At any rate, it is definitely indicated prior to any consideration of terminating the pregnancy. Also, vitamin B_1, by its timely use, should prove of value in the prevention of neuronitis of pregnancy.

The successful treatment of neuronitis of pregnancy by vitamin B_1 might be more prevalent if we followed the lines of Goodhart and Jolliffe.[13] They found in the treatment of polyneuritis of alcohol addicts, that the best results were obtained following the intravenous injection of large doses of natural or synthetic crystalline vitamin B_1. It is possible that inadequate vitamin B_1 therapy might, in some instances, have accounted for the unsuccessful results reported in the treatment of neuronitis of pregnancy.

CASE REPORT

M. J., 25 years of age, a white American primigravida, had been married three years.

The family history was unimportant. Her father had died of carcinoma of the liver at the age of 37, and a brother had died following infantile paralysis at the age of 2.

The past personal history revealed no previous illnesses or operations other than a tonsillectomy in 1922. The menses began at 14, recurred regularly every twenty-eight days, were of three days' duration, and were not associated with pain or the passage of clots. The last period began April 15, 1937.

The patient started to vomit about three weeks after this period and continued to do so for eight weeks. At the outset vomiting was marked in amount and frequency, and was soon accompanied by considerable loss of weight and weakness. The weight dropped from 208 to 152 pounds. Drowsiness appeared and became more pronounced as time went on. The patient's legs became especially weak and numb, and there was a feeling of numbness in the hands. She was finally unable to stand or feed herself.

Her physician suspected a toxic neuronitis of pregnancy and began the intramuscular administration of vitamin B_1 in doses of 2,000 I.U. daily, in addition to estrin, glucose intravenously, sedatives, and liver extract. In spite of this therapy, the weakness, numbness and drowsiness increased progressively, and, in addition, loss of motion of both hands appeared.

The patient was admitted to the Prospect Heights Hospital August 19, 1937, in fair general condition. She complained of pain in her feet, legs and abdomen, inability to stand, loss of use of the hands, and numbness of the hands and feet.

On physical examination, the blood pressure was 144/96. The heart and lungs were essentially normal except for tachycardia with an apex rate of 124 per minute. The abdomen was tender in the right lower quadrant. The liver and spleen were not palpable. The uterus reached to within three fingerbreadths of the umbilicus. No fetal heart or movements could be heard.

The eyes showed a lateral and vertical nystagmus. The pupils reacted well to light and accommodation. There was loss of touch sensation in both lower extremities and in the abdominal wall up to the sternum. Pain sensation (pinprick) was impaired in both lower extremities and in the abdominal wall. The tendon reflexes were present in the upper extremities, but absent in the lower. There was also a bilateral wrist-drop and foot-drop.

Pelvic examination disclosed a nulliparous introitus, and a soft, closed, insensitive cervix situated in the axis of the vagina. The fundus was the size of a four and one-half to five month gestation; it was smooth and soft and lay in the midline. The fornices were normal.

The patient cried because of pain in the abdomen, feet and legs. She was irrational at times and tried to get out of bed. There was periodic incontinence of urine and feces. Her conversation was rambling and incoherent at times, and there was a tendency toward talkativeness. She vomited occasionally and would eat only after a great deal of coaxing.

Treatment consisted of vitamin B_1 (2,000 I.U. daily), intravenous glucose, combined vitamins and minerals (vi-syneral), sodium luminal, sodium amytal, strontium bromide, liver extract, sodium bicarbonate enemas, and light plaster casts for ankle immobilization.

She improved generally and was happy in the fact that her feet felt warmer than they had in several weeks. She finally insisted upon leaving the hospital, which she did, by ambulance, ten days following her admission.

The temperature and respirations were within normal range throughout the hospital stay, but the pulse varied between 90 and 125, similar to the rate prior to her admission to the hospital.

The patient was re-admitted on August 31, after having remained at home for two days. During that period she was irrational most of

the time, her mental status became worse, and she refused all food, although she was not vomiting. On her second admission to the hospital, she was seen by Dr. A. M. Rabiner, and the following neurologic opinion was rendered: "The patient is conscious, coöperative, and jovial. She states that she is neither happy nor unhappy. She is well oriented except for the day of the week, and has no recollection of having created any disturbance in the hospital or at home yesterday.

"There is a horizontal nystagmus to both the left and the right. Other ocular movements are normal and there is no fundal pathology. There is a bilateral wrist-drop and foot-drop with weakness in all four extremities, most marked distally, with, however, preservation of the supinator longus muscles. The tendon reflexes are active in the upper extremities, but only the right ankle jerk is obtained in the lowers. The abdominal reflexes are present but diminished. There is extreme muscle tenderness. There is sensory impairment for all forms maximally in the distal parts with a level at about the thoracic sixth on the chest. This is particularly so for touch, but vibration is not felt below the clavicles. There are patchy areas of preserved sensation over the lower extremities and, at times, retardation in conduction time can be demonstrated. There are no pyramidal tract signs except for the hyperactive arm reflexes.

"Opinion: The history of pernicious vomiting for a prolonged period with loss of weight (more than 50 pounds), and thereafter the development of a polyneuritis and how mental symptoms, warrants the diagnosis of a diffuse neuronitis involving the peripheral nerves, the cord, and even the brain. On the basis of a metabolic toxic disorder induced by the underlying pregnancy, the examiner feels that with the increasing symptomatology, despite all attempts at therapy, including vitamins, liver, etc., the pregnancy should be interrupted."

On September 1, 1937, a vaginal hysterotomy was done under rectal avertin anesthesia and open drop ether. The fetus was 28 cm. long and of male sex. The placenta was 8 cm. in diameter and grossly normal. The patient's condition was poor throughout the operative procedure. The pulse ranged between 152 and 160. Coramine was used in addition to glucose intravenously. The postoperative course was quite stormy. There was immediate postoperative shock, with a drop in blood pressure to 80/60. She reacted well, however, to transfusion and general shock treatment.

She was incontinent, irrational and uncoöperative, crying and shouting in a manner which disturbed the other patients in the hospital. She forgot her name, was disoriented as to time or place, and when rational, she would fret about her loss of memory and the possibility that she might never be able to walk. Her physical improvement was more rapid than her mental. By the sixteenth postoperative day the condition of her hands had improved sufficiently so that she was able to pick up paper napkins without difficulty. Also, at this time she was completely continent and mentally clear. She was discharged from the hospital on September 19, seventeen days postoperative.

The treatment during her second admission, in addition to termination of pregnancy, included vitamin B₁, liver extract, codeine, chloral hydrate, sodium bromide, hyoscine-morphine-cactoid, tincture of hyoscyamus, blood transfusion, and bladder instillations of argyrol.

The temperature was 100°F. on admission and reached normal prior to operation. Postoperatively, there was a climb to 105.6 within ten hours, with a gradual fall to 99 in the following thirty-six hours, followed by a rise to 103. The temperature finally tapered down to normal on the ninth postoperative day, and remained normal up to the time of discharge from the hospital. The pulse ranged from 90 to 120 preoperatively, but reached 156 postoperatively. It gradually slowed down, but never went below 100, which it was at the time of discharge from the hospital. The respirations were not affected except immediately following the hysterotomy, when they reached 40 and remained at that level for several hours.

Urine analysis was normal except for traces of albumin and the presence of clumps of pus. The specific gravity varied from 1.010 to 1.023.

Blood chemistry:

Sugar	100 mg.
Urea nitrogen	15 mg.
Uric acid	4.5 mg.
Creatinine	1.5 mg.
Chloride	503 mg.
Phosphorus	4.34 mg.
Calcium	10 mg.
Blood grouping	0
Kline precipitation test	Negative

The blood count showed red blood cells varying from 3,780,000 to 4,150,000, hemoglobin from 60 to 67 per cent, white blood cells from 5,850 to 11,100, neutrophiles from 54 to 72 per cent, and lymphocytes from 20 to 28 per cent. The color index was 0.8.

Periodic examinations following her discharge from the hospital showed gradual improvement in all symptoms. When last seen during January, 1939, the patient stated that she felt well and was able to perform all her daily duties without physical or mental handicap. Physical examination at that time revealed her to be normal except for sluggish patellar reflexes.

SUMMARY AND CONCLUSIONS

A case of neuronitis following pernicious vomiting of pregnancy is presented.

Vitamin B_1 therapy did not check the progress of the disease. Termination of pregnancy was carried out by vaginal hysterotomy. This was followed by prompt alleviation of symptoms and a gradual, progressive return to normal, which has continued up to the time of this report.

Neither vitamin B_1 therapy nor interruption of pregnancy alone is the complete answer to the problem of toxic neuronitis of pregnancy. A trial of the former in large doses should always be attempted before resorting to therapeutic abortion. Termination of pregnancy may be necessary in addition to vitamin B_1 therapy where the latter does not produce a promising response in a reasonable time.

It is emphasized that vitamin B_1 therapy be employed as a prophylactic agent in all cases of pernicious vomiting of pregnancy, regardless of whether symptoms suggestive of an approaching neuronitis are present or not. It is only by the observance of this precaution that the occasional case which may develop will be prevented.

REFERENCES

1. BERKWITZ, N. J., and LUFKIN, N. H. *Surg., Gynec. & Obst.*, 54: 743, 1932.
2. STRAUSS, M. B., and MCDONALD, W. J. *J. A. M. A.*, 100: 1320, 1933.
3. MAISEL, J. J., and WOLTMAN, H. W. *J. A. M. A.*, 103: 1930, 1934.
4. LUBIN, S. *Am. J. Obst. & Gynec.*, 26: 442, 1933.
5. PLASS, E. D., and MENGERT, W. F. *J. A. M. A.*, 101: 2020, 1933.
6. VANDEL, D. T. *J. Missouri State M. A.*, 30: 407, 1933.
7. THEOBALD, G. W. *Lancet*, 1: 834, 1936.
8. FOUTS, P. J., GUSTAVSON, G. W., and ZERFAS, L. G. *Am. J. Obst. & Gynec.*, 28: 902, 1934.
9. COOK, B. A. *M. J. Australia*, 2: 319, 1935.
10. STROH, J. E. *Northwest. M. J.*, 33: 360, 1934.
11. BINGHAM, A. W. *Am. J. Obst. & Gynec.*, 32: 144, 1936.
12. FORMAN, G. W. *J. Missouri State M. A.*, 34: 197, 1937.
13. GOODHART, R., and JOLLIFFE, N. *J. A. M. A.*, 110: 414–419, Feb., 1938.

A CASE OF PURE TRAUMATIC RUPTURE

WALTHER EHALT, M.D.

Chief Assistant, Unfallkrankenhaus

VIENNA, AUSTRIA

PURE traumatic ruptures are extremely rare and always occur by direct trauma. They are true accident

We present here briefly a case which was observed at the Unfallkrankenhaus in Vienna:

FIG 1.

injuries and therefore entitled to compensation. For their recognition a definite direct trauma is necessary, with all symptoms,

An 18 year old laborer riding on his bicycle was hit by a wagon tongue on the right side of his abdomen and was thrown from his bi-

FIG. 2.

such as swelling, hematoma, tenderness and sometimes skin abrasions over the injured place. Also of importance is the finding of torn musculature at time of operation and possibly also the histologic report.

cycle. He was a robust, well-nourished man in good general condition, with normal temperature and pulse of 80. No vomitus or nausea was present. The right abdomen revealed a tumor the size of two men's fists at the level of the umbilicus at the outer margin of the rectus

abdominus. There were skin excoriations 1.5 cm. in diameter. Gut could be palpated under the skin corresponding to the tumor and could be reduced through a hole in the abdominal wall of 4 fingerbreadths. Bed rest was advised for about ten days until the excoriation was healed and most of the hematoma was absorbed. The peristalsis was sluggish during this time so that cathartics had to be given. Pain was present only upon coughing.

Operation was done under ether anesthesia. An oblique incision 15 cm. in length was made over the rupture. After the skin incision a cavity the size of a fist, with smooth epithelial lining and filled with gut and omentum, was opened. The posterior wall of the cavity was formed by the anterior abdominal fascia in which there was a hole 5 cm. in diameter. The dimensions of the cavity were 8 cm. The upper margin of the entrance to the abdominal cavity was free. There were adhesions of omentum and gut to the lower margin. The adhesions could be easily loosened. The peritoneum was incised and freed and closed. The muscle layers were then dissected. The rectus abdominus muscle was normal. The internal oblique was torn for about 8 cm. The external oblique showed a longitudinal tear. The muscles were closed in layers and the anterior rectus sheath was closed. Subcutis and skin sutures were placed and a subcutaneous rubber drain was inserted for twenty-four hours.

Healing occurred by primary union. The patient was discharged as cured thirty-two days following the accident. One year later he was completely recovered. (Fig. 2.) He works as well as before the accident, his digestion is normal. The scar is solid, smooth and mobile.

The histologic findings of the specimen removed from the anterior wall of the sac showed fatty tissue which gradually blended towards the inner surface of the sac into looser connective tissue, with extensive free hemorrhage in the connective tissue. The superficial layers revealed coagulated blood with positive fibrine reaction and beginning organization. The surface was covered by endothelium. The fatty tissue showed microblasts and a few lymphocytes.

The case demonstrated all necessary signs of a recent injury clinically and surgically. Such pure cases are very rare. As a rule patients with an inguinal hernia come for medical observation stating that they had a sudden severe pain lifting a heavy load. These cases are commonly congenital inguinal hernias. Trauma is only the determining factor of the rupture which itself is preformed. Such cases have to be rejected for compensation.

AN UNUSUAL CASE OF PARALYTIC ILEUS FOLLOWING REMOVAL OF CATARACT

EDWARD J. KILFOY, M.D.

Surgical Staff, Queen of Angels, St. Vincent's and Cedars of Lebanon Hospitals

LOS ANGELES, CALIFORNIA

PARALYTIC ileus is a common complication following intra-abdominal surgical procedures, but ileus following the removal of a cataract under local anesthesia is unusual. Whether the removal of the cataract in the case here reported had anything to do with the production of ileus is problematic. Although volume upon volume has been written on the subject of ileus, and although much experimental work has been done, its etiology remains obscure. It is doubtful whether adynamic ileus, as induced in the experimental animal, is the same as the form which occurs in the human.

CASE REPORT

R. B., a dentist, aged 54, married, reported for examination because of progressive loss of vision in the right eye of about two and one-half years' duration. For the previous three or four months there had been gradual loss of vision in the left eye as well.

The previous history was negative except for excessive usage of alcoholic beverages, dating to student days. However, the patient was a moderate smoker and did not use drugs. He had had no serious illnesses, operations or accidents.

Physical examination showed that he weighed 190 pounds, had a blood pressure of 150/84, pulse of 78, respirations 17 and temperature of 98 degrees. A cataract covered the entire right pupil. The patient could not see fingers or print with the right eye at any distance, and although he could see fingers and differentiate numbers with the left eye, he stated that they did not appear clear. The abdomen was somewhat pendulous, but there was no tenderness, rigidity, or tumor mass. Liver, spleen and kidneys were not palpable. In other respects, the examination proved negative.

The urine was cloudy, of alkaline reaction, with a specific gravity of 1.017. No sugar or acetone was present. but there was a very faint trace of albumin. Occasional leucocytes were observed, but no red cells or casts.

The red blood cells numbered 5,880,000 and the leucocytes 9,000, with polynuclears 69 per cent, lymphocytes 24 per cent, large monocytes 7 per cent. The hemoglobin was 92 per cent and the color index 0.81. Blood Wassermann proved negative.

A diagnosis of bilateral cataract was made and the patient referred for operation.

Operation. Under cocaine and novocaine anesthesia the right cataract was removed on December 17, 1934. Iridectomy was done through a limbus incision, with the formation of a conjunctival flap. The anterior capsule was removed with capsule forceps and the nucleus and cortical masses expressed. The patient was returned to bed in good shape.

Two days after operation, however, he began to have abdominal pains and distention, although temperature and pulse remained good. Gastric lavage yielded nothing. Rectal tube, enemas and limitation of oral fluid intake were recommended to control the dilatation. By December 21, the upper half of the abdomen was greatly distended, tympanitic and tender on palpation. Some gas passed per rectum and through the rectal tube and large amounts of gas were belched. The leucocytes numbered 7,000, and temperature and pulse were normal. After gastric lavage, a nasal tube was inserted for continuation of the lavage. Ten per cent glucose in normal saline, 1,000 c.c., was given intravenously.

Intravenous glucose was continued, but on December 23, two days later, the patient became irrational, and pulled out the nasal catheter. Pitressin, hot abdominal stupes, enemae, a rectal tube and diathermy were prescribed, but abdominal distention increased. On the following day the pulse was rapid and weaker, the temperature had risen to 101

degrees. The bowels had moved involuntarily several times during the night. The blood pressure was 106/76; the pulse 116. On the next day, after a very restless night, the patient was semi-comatose, with rapid, weak pulse and definite evidence of myocardial failure. At 5:40 A.M. he died. Convalescence from the cataract operation, as shown by the oculist's notes, had been satisfactory.

Post-Mortem Examination. Autopsy was limited to the abdomen. Dense adhesions between the greater omentum and the brim of the pelvis, as well as to the abdominal wall in the region of the cecum and appendix, were apparent upon opening the peritoneal cavity. The loops of small bowel were greatly distended; there was moderate distention of the ascending colon. The peritoneum had lost its glistening appearance, but there was no exudate. No areas of obstruction were found in the gastrointestinal tract. The liver weighed 1,800 Gm., and showed large, irregular, yellowish patches—areas of necrosis.

An anatomic diagnosis of adynamic ileus, toxic necrosis of the liver and old plastic peritonitis in the region of the appendix was reported.

Microscopic sections of the liver showed extensive necrosis of the type seen in chloroform poisoning, with vacuolization of the liver cells and interstitial hemorrhage.

COMMENT

This case of cataract had adynamic ileus and toxic necrosis of the liver as major complications. The ileus involved the small bowel. The acute toxic necrosis produced a destruction of approximately two-thirds of the liver substance, which was replaced by fat.

If at the end of twenty-four to thirty-six hours, regardless of the surgical or mechanical procedure carried out, the patient begins to have abdominal distention and tenderness, unrelieved by routine measures, one must think of adynamic ileus. The mortality in this type of case is very high.

Just what reflex mechanism took place, if any, in the removal of the cataract that produced the ileus, I am not able to state. I doubt very much if the cataract removal had anything to do with the ileus. The reaction of the long and continued use of alcoholic beverages on the patient's liver had probably changed its physiologic-pathologic condition so that it was not able to handle the toxins produced from the bowel obstruction. There is also the question of the distention of the bowel, crowding the liver and interfering with circulation of the right hepatic artery. This may have produced a thrombus with subsequent necrosis of the liver. However, this is questionable, as both the right and left lobes of the liver were involved to the same extent. Also at autopsy, there were no findings in the hepatic arteries.

This man might well have developed adynamic ileus no matter what type of operation or mechanical manipulation had been carried out.

SPONTANEOUS URETERAL RUPTURE WITH PERI-URETERAL ABSCESS FORMATION*

SIDNEY SMITH, M.D.

RALEIGH, NORTH CAROLINA

IN a survey of the literature few case reports of non-traumatic rupture of the ureter could be found. An impacted ureteral stone followed by the usual secondary inflammatory process resulting in narrowing of the ureter was the cause of the rupture in every case. Surraco[1] reports eight such cases and Rathbun[2] describes a similar case in which there was rather marked necrosis of the ureter with abscess formation due to blockage from a ureteral stone. Edgar Freshman[3] reports a case of spontaneous rupture of the ureter and adds it to eleven others found in the literature, all of which were secondary to stone.

Clinically these cases are characterized by a history of sudden onset of severe cramping pain localized in the abdomen and sometimes radiating to the flank. There is marked rigidity of the abdominal muscles on one side, which may extend to the opposite side. These abdominal symptoms are not present in rupture of the parenchyma of the kidney because the process extends to the outside and behind. In ureteral extravasation there is direct contact with the peritoneum and the resulting abdominal signs may be confused with an intra-abdominal lesion. The patient is usually quite ill with high fever, weakness, nausea and vomiting. Most striking of all is the marked scarcity of urinary symptoms.

The following case is presented, first because of its rather unusual occurrence and second, because a ureteral stone could not be demonstrated as a causative factor in the rupture. The postoperative study is also a striking illustration of the recuperative power of the kidney and ureter.

CASE REPORT

The patient was a white male, age 25, who was admitted to the surgical service of Rex

FIG. 1. Plain urinary tract x-ray showing scoliosis, absence of left psoas muscle outline and a dense shadow indicating mass below and medial to left kidney shadow.

Hospital on May 7, 1937. The first symptoms noticed were general malaise, vague abdominal pains, nausea, vomiting and high fever, all of which began two weeks prior to his admission. On the following day the pain became very sharp and cramping in character. It was localized chiefly in the left lower abdominal quadrant but radiating also to the left flank and right lower quadrant of the abdomen. His family physician was consulted and a tentative diagnosis of appendicitis with possible peritonitis was made. There was no history of any urinary symptoms.

Clinical Examination. The patient appeared quite ill and his facial expression was

that of one in much pain. Temperature was 101°F.; pulse 100; blood pressure 125 systolic and 80 diastolic. The tongue was heavily coated

FIG. 2. Left urogram. Dilatation of pelvis and upper ureter with rupture of ureter and extravasation of skiodan solution.

and the tonsils were enlarged and badly infected. There were no abnormal chest findings. The abdomen was flat and symmetrical. The muscles of the middle and lower abdomen were quite rigid and there was exquisite tenderness in both lower quadrants. Neither flank showed any sign of swelling but deep pressure in the left flank elicited slight pain. Rectal examination revealed a normal prostate gland.

Urine examination showed an occasional pus cell and a slight trace of albumin. The white blood cell count was 27,200 with 90 per cent polymorphonuclear leucocytes; red cell count was 4,000,000; hemoglobin 70 per cent; the Wassermann was negative.

Urologic Study. On admission the tentative diagnosis was an acute abdomen, but after observation the patient was transferred to the urologic service. A plain urinary tract x-ray showed the right kidney shadow slightly larger than the left. The outline of the right psoas muscle was easily defined while the left was completely obscured. There was a shadow suggestive of a mass about the size of a large orange just below and medial to the lower pole of the left kidney. (Fig. 1.) Cystoscopy revealed a

normal bladder except for slight congestion around the left ureteral orifice. Indigocarmine, 5 c.c., was given intravenously and appeared

FIG. 3. Postoperative urogram. Pelvis almost normal. Marked narrowing of ureter at point of rupture.

from the right ureter in four minutes in good concentration, but none came from the left in fifteen minutes. The left renal pelvis was easily catheterized but no urine could be obtained. A left urogram, using 15 per cent skiodan solution, showed considerable dilatation of the calyces and pelvis. Five cm. below the ureteropelvic junction there was a point of ureteral rupture with extravasation of the skiodan into a large cavity which lay medial to the catheter and measured 4 × 8 cm. in size. (Fig. 2.)

On the following day, under gas anesthesia, a left curved anterior kidney incision exposed a large peri-ureteral abscess containing about 200 c.c. of thick, foul pus which was loaded with colon bacilli. The cavity extended from the inner margin of the lower pole of the left kidney downward for a distance of approximately 10 cm. and was in direct contact with the peritoneum which had been pulled far back posteriorly. The convalescence of the patient was uneventful and a striking observation was the fact that no urine drained from the incision until two weeks after the operation. This continued for one week and the wound then healed rapidly.

Postoperative Study. Cystoscopy showed the left kidney function to be good. There was little abnormal change in the renal pelvis. The minor calyces were well defined. At the point of ureteral rupture the outline is irregular, slightly tortuous for a distance of 2 cm. (Fig. 3.)

SUMMARY

A case of non-traumatic rupture of the ureter with periureteral abscess formation not secondary to ureteral stone is presented. The unusual feature of this case is the absence of all urinary symptoms or physical findings which might lead to a diagnosis of renal or ureteral pathology.

REFERENCES

1. Surraco. Spontaneous pyelo-ureteral rupture. *J. d'urol.*, Jan., 1937.
2. Rathbun, N. P. Necrosis of the ureter, perforation, peri-ureteral abscess. *J. Urol.*, 18: 329 (March) 1927.
3. Freshman, Edgar. Extravasation of urine following spontaneous rupture of the uretero-pelvic junction. *Brit. J. Urol.*, 7: 267 (Sept.) 1935.
4. Abeshouse, B. Rupture of kidney pelvis. *Surg., Gynec. & Obst.*, 60: 710, 1935.
5. Hamer, G. L. Practice of Surgery, Urology. Volume 8, chapter 2.

The incidence of stones in hyperparathyroidism is said to be 70 per cent and 5 per cent of all cases of nephrolithiasis are alleged to be due to parathyroid disease.

From—"The Endocrine Glands," by Max A. Goldzieher (D. Appleton-Century).

CHRONIC INVAGINATION OF MECKEL'S DIVERTICULUM

GEORGE H. BUNCH, M.D.

COLUMBIA, SOUTH CAROLINA

OVER a hundred years ago Johann Meckel found that a part of the vitalin duct may persist as a con-

FIG. 1. Section of unopened ileum. A, base of invaginated diverticulum; B, distal end of invaginated diverticulum; C, site of omental adhesion.

gential diverticulum of the small intestine. The anomaly, known as Meckel's diverticulum, has long been recognized as being prone to acute inflammation and to perforation with clinical manifestations and laboratory findings almost identical with those of acute appendicitis. If its distal end becomes adherent to the abdominal wall or to a viscus the diverticulum acts as a band which may mechanically obstruct the intestine or be the cause of volvulus.

Since Denecke in 1902 found an ulcer in a diverticulum and Deetz in 1907 found an area of mucosa in a diverticulum, microscopically identical with gastric mucosa, our concept of Meckel's diverticulum has considerably changed. We now know that heterotopic gastric mucosa there has the characteristics of gastric mucosa in the stomach. They both secrete hydrochloric acid and both tend to develop peptic ulcer complicated by hemorrhage and perforation. In rectal bleeding at any age the source may be a diverticular ulcer. When no local lesion is found on proctoscopic examination and no stomach or duodenal ulcer found on x-ray study, a bleeding diverticular ulcer should be suspected, especially in children. The hemorrhage may be so profuse as to be the direct cause of death.

A growing literature based on laparotomy and necropsy findings on a scale never before available proves that as a cause of disease the ubiquitous Meckel's diverticulum may play a surprisingly varied rôle. It may be the site of tumors both malignant and benign. Carcinoid tumor, adenoma, adenocarcinoma and several types of sarcoma have been reported.

By becoming invaginated into the lumen of the intestine, as a finger of a glove may be inverted, it is now known to be a cause of acute intussusception. The percentage of cases of Meckel's diverticulum causing intussusception is not known, but it is thought that 1 per cent of all cases of intussusception are due to it. DeQuervain thinks that invagination comes from suction in the intestine caused by the vacuum produced by rapid passage of its contents. Lower thinks it may be due to peristalsis of the diverticular walls in the effort to expel its contents or, "and this is by far the most accepted opinion, to an inflammatory process about the opening of the diverticulum which produces thickening of its walls, this process gradually extending so that the mucous membrane at the opening is slowly pushed along with the peristaltic action and the contents of the intestine."

The symptoms vary with the pathology. If invagination of the diverticulum is immediately followed by intussusception of the intestine the symptoms are those of acute intussusception—severe pain followed by nausea and shock. Rigidity and local tenderness are slight. Palpable tumor may or may not be present. Bloody stools have been observed although Lower stresses their absence. When intussusception does not immediately follow invagination there

are usually spells of recurrent intestinal colic. That invagination of a diverticulum may persist for an indefinite time without striction and extending distally, within the lumen of the intestine, about 2 inches. There was no evidence of inflammation. Not knowing

FIG. 2. Invaginated diverticulum delivered through opening in ileum.

FIG 3 Ileum opened showing invaginated diverticulum.

causing intussusception or incapacity in the patient is proved by the following case:

Mrs. R. H. H., white, age 25, para 3, was admitted to the South Carolina Baptist Hospital August 27, 1937 for the relief of uterine bleeding present off and on for several months after miscarriage. Next day, after curettage had been done with negative findings, a midline incision was made and the uterus was found to be retroverted and adherent, with both adnexa normal. The uterus, the cervix and the appendix were removed. In placing the omentum over the viscera before closing the incision a tag of omentum was found adherent to the small intestine about 3 feet proximal to the ileocecal junction. In freeing this a constriction of the intestine was found about 2 inches distal to the omental adhesion and from the constriction a flat mass was felt attached to the intestine at the place of con-

what the condition might be, we removed the involved segment and did a side-to-side anastomosis of the ileum. Convalescence was uneventful and the patient was discharged on the twenty-first day.

The report of Dr. Kenneth Lynch, Professor of Pathology of the Medical College of South Carolina, on the specimen is:

"Received a section of small intestine 20 cm. in length. Towards one end there is an accumulation of mesentery on the outer surface, opposite the internal lesion to be described. Projecting into the lumen of the bowel at this point is a blunt curved mass 4.5 × 1.5 × 1.5 cm. This mass is attached to the intestinal mucosa at its base and is itself covered with mucosa. It contains a mass of fat continuous with that of the mesentery and the gross impression is that the intestinal wall at this point has been invaginated and the invagination filled with

mesentery. The adjoining intestinal tract appears fairly normal.

"From gross and microscopic appearances, this is interpreted as probably an inverted diverticulum although it may be an intussusception of mesenteric tissue into the intestinal wall. There is no appearance of any part of it having sloughed away."

COMMENT

This is a case of chronic invagination of a Meckel's diverticulum found incidentally at laparotomy for a gynecologic condition which could in no way have been related to the intestinal lesion. When the invagination occurred and how long it had persisted cannot be told. An attempt at reduction of the invagination after resection failed, for the serosa of the diverticulum was fixed in inversion by fibrous adhesions. A review of the patient's history taken on admission showed no symptom referable to the diverticulum and when questioned after operation she did not recall ever having had an acute attack of abdominal pain which might have marked the time of invagination. This, with the absence of recurring attacks of intestinal colic, makes one think that the diverticulum may have become invaginated and continued that way for an unknown time without causing symptoms of sufficient degree to have been remembered.

REFERENCES

MECKEL, J. F. Handbuch der pathologischen Anatomie. 1: 555–597, Leipzig, 1812–1818.

DENECKE. Ueber die Entzuendung des Meckel Divertikels und die Gangraen desselben. *Deutsche Ztschr. f. Chir.*, 62: 523, 1902.

DEETZ, E. Perforationsperitonitis von einem Darmdivertikel mit Magenschleimhautbau ausgehend. *Deutsche Ztschr. f. Chir.*, 88: 482, 1907.

COBB, D. B. Meckel's diverticulum with peptic ulcer. *Ann. Surg.*, pp. 130–147, 1935.

LOWER, WM. E. Intussusception in adults due to the invagination of a Meckel's diverticulum. *Ann. Surg.*, 82: 436, 1925.

GRAY, H. K., and KERNOHAN, J. W. Meckel's diverticulum associated with intussusception and adenocarcinoma of ectopic gastric mucosa. Report of a case. Collected Papers of the Mayo Clinic and the Mayo Foundation, 28: 166, 1936.

CHESTERMAN, J. T. Hemorrhage per rectum as an indication of disease in a Meckel's diverticulum. *Brit. J. Surg.*, 23: 267, 1935.

McKECHNIE and PRIESTLEY. Intussusception of Meckel's diverticulum. *Proc. Staff Meet. Mayo Clin.*, 2: 545–544 (Aug. 19) 1936.

HARKINS, HENRY. Intussusception due to invaginated Meckel's diverticulum. Report of 2 cases with a study of 160 cases collected from the literature. *Ann. Surg.*, 98: 1070–1095 (Dec.) 1933.

DRUMMOND, H. Inversion of Meckel's diverticulum. *Ann. Surg.*, 55: 402–415 (Mar.) 1912.

EISBERG, H. B. Inversion of tumor of Meckel's diverticulum in a double ileocecal intussusception. *Am. J. Surg.*, 5: 401–404 (Oct.) 1928.

KLINGENSTEIN, P. Enteric intussusception due to the invagination of Meckel's diverticulum. *J. Mt. Sinai Hosp.*, 3: 1–4 (May-June) 1936.

ALLMAN, D. B. Meckel's diverticulum as the cause of intussusception. *J. Med. Soc. New Jersey*, 25: 352–354 (May) 1926.

CANNON, D. J. Double intussusception due to Meckel's diverticulum. *Irish J. M. Sc.*, Series 25, p. 27 (Jan.) 1928.

REISER, E. Invagination of the small intestines due to a Meckel diverticulum. *Röntgenpraxis*, 7: 90–94 (Feb.) 1935.

DE LANGRE, M. Rare type of intestinal invagination due to Meckel's diverticulum. *Bull. Soc. nat. de chir.*, 58: 925–927 (June 25) 1932.

MATHIEU, P. and DAVIOUD. Intestinal invagination of Meckel's diverticulum. Resection of the intestine. *Bull. Soc. de pediat. de Paris*, 25: 132–133, 1927.

BETTAZZI, G. Intussusception caused by Meckel's diverticulum; clinical report of a case. *Bull. et mém. Soc. nat. de chir.*, 58: 925–927 (June 25) 1932.

KORIN, P. I. Invagination of Meckel's diverticulum. *Vestnik khir.*, 46: 61, 1936.

PULMONARY EMBOLI FOLLOWING INJECTION OF VARICOSE VEIN

CONTROL BY LIGATION OF EXTERNAL ILIAC VEIN

K. P. A. TAYLOR, M.D., F.A.C.S.

HAVANA, CUBA

RECENT emphasis of the need for high ligation of the saphenous vein in conjunction with its injection[1] will attain an end unmentioned by its proponents, namely, elimination of pulmonary embolism. This most hazardous complication of the injection treatment of varicose veins is generally disregarded in current literature. Since accurate clinical means of identifying individuals of thrombotic tendency are lacking,[2] embolism may occur in a quite uncomplicated and propitious case, inducing in patient and physician alike a most acute anxiety. This sense of impending fatality may be mitigated by indicating the feasibility of placing a mechanical barrier above the site of thrombosis.

If one is fortunate enough to operate before the thrombus has mounted to the vena cava, ligation of the external iliac vein (possibly the common iliac vein) will accomplish this end. The timely identification of the iliac and femoral veins as the most frequent sites of postoperative thrombophlebitis[3] suggests the possibility of utilizing this operation as a prophylactic against embolism in these threatening cases.

It should also be emphasized that pulmonary embolism may follow the use of one of a group of drugs known at times to produce severe anaphylactic reactions,[4] but generally considered incapable of causing embolism—in this instance, sodium morrhuate.

CASE REPORT

A. G., white, age 34, a radio operator on the S.S. Virginia, was seen in consultation with Chief Ship's Surgeon Josephs on August 11, 1936. On the preceding day, the patient had experienced an excruciating pain in the right side of his chest, spat up a large amount of bright blood, and collapsed. Six days previously he had a single (and first) injection of sodium morrhuate (5 c.c. of 5 per cent solution) in the middle third of his right saphenous vein. (Correspondence with the physician who gave this injection affirms that the vein was varicosed grade two, but not inflamed, i.e., that no contraindication existed.) The injection site and vein became moderately tender, but characteristically gave no indication of embolic potentiality.

The patient had consolidation of the right lower and middle lobes; he was dyspneic and cyanotic, with continuous hemoptysis of fresh blood; temperature was 102, pulse 110, respirations 34. He was admitted to the Anglo-American Hospital, Havana, with the diagnosis of lobar pneumonia following massive pulmonary embolism. The blood examination disclosed: erythrocytes 3,700,000; hemoglobin 75 per cent; leucocytes 16,400; differential: segmented neutrophiles 76 per cent; stabs 10 per cent; lymphocytes 14 per cent. Thromboplastin and calcium lactate were given and rapid digitalization commenced. Twenty c.c. of polyvalent antipneumococcus serum was injected intramuscularly.

On the following day, 60 c.c. of antipneumococcic serum was given intravenously. Pneumothorax of 200 c.c. was induced. The sputum yielded staphylococci and pneumococci Type 3.

On the third day, the pneumothorax was increased by the addition of 400 c.c. Following this, the patient was comfortable, and his temperature declined to normal on the sixth hospital day (seventh day after embolism). Urinalysis was essentially normal, the Kahn test negative, and repeated sputum examinations failed to disclose tubercle bacilli. At no time was the right leg swollen or painful.

On the seventeenth day of illness, a knife-like pain in the left side of the chest and hemoptysis heralded a second embolism. The infarcted area was in this instance comparatively small

and caused little distress. The patient and his attendants were now thoroughly alarmed. The advisability of ligation above the thrombosed

Fig. 1. Absence of edema of right leg following ligation of external iliac vein.

area was evident. Accordingly, on the third day following the second embolism, spinal anesthesia (procaine hydrochloride 0.15 Gm.) was administered. The common femoral vein was exposed immediately below the inguinal ligament. It was incompletely thrombosed and softened; the first catgut ligature penetrated the vessel—a soft purple clot and a small amount of fresh blood exuded. Section of the vein was completed between additional ligatures. The saphenous vein was also ligated at its juncture with the femoral. An attempt was made to dissect the proximal end of the femoral vein beneath the ligament, but this was hazardous because of scarring inflicted by an old hernia operation. The incision was therefore closed.

At this time the plasma clotting time was 7½ minutes initial and 8¾ minutes complete. The basal metabolic rate was minus 9 per cent. Thyroid extract 1 gr. t.i.d. had been given since the second embolism; in addition, sodium thiosulphate 20 c.c. of 10 per cent solution, had been injected daily intravenously. This intelli-

gent patient was told that the probability of having a third embolism had been lessened by the femoral vein ligation, but not entirely removed. He thereupon elected to have the external iliac vein exposed.

The abdomen was opened, again under spinal anesthesia, five days after the first operation. A Battle incision was used. Difficulty was encountered in identifying the vein, since it was smaller and harder than the artery, and partly collapsed. It was ligated above the deep epigastric and deep circumflex iliac branches with two strands of No. 4 chromic catgut, passed beneath the vein with a ligature carrier. The ligatures were carefully secured one-fourth inch apart. The vein was not divided.

Following this operation, assurance was felt and given that further embolism could not occur. The administration of thyroid extract and sodium thiosulphate was stopped, tonics were ordered and a liberal diet supplied. Ten days after ligation of the right external iliac vein, afebrile inflammation of the *left saphenous* vein occurred. This was accompanied by moderate swelling of the left leg and tenderness in the calf. The plasma clotting times were now four and six minutes, a situation disposing to thrombosis, perhaps embolism. A relationship between this left-sided phlebitis and previous blockade of the right external iliac is of course admissible.

Sodium thiosulphate and urotropin, intravenously, were employed to combat thrombosis and phlebitis. The inflammation subsided in ten days, and the patient was discharged, well, on October 8. At this time slight edema of the right calf and ankle followed several hours of walking. Following ligation of the vein, no swelling had occurred.

TECHNIQUE OF LIGATION

It is doubtful whether this operation can be confidently performed without spinal anesthesia. Careful incision of the posterior parietal peritoneum, separation of the artery and vein from their common sheath and from each other, and passing of the ligature carrier before introducing the ligature are important features. The choice of material may depend upon the vein's condition, whether indurated or softened, dilated or collapsed; likewise the method of ligation may be the operator's choice.

Slicing of the vein is of course the prominent hazard; on the other hand, failure fully to close the vein would be worse than non-operation, since partial ligation would itself initiate a creeping thrombus.

SUMMARY

1. Because of the dangers of thrombosis and embolism, high ligation of the saphenous vein should probably be done in *all* varicose veins to be injected.

2. Following embolism of both lungs, ligation of the external iliac vein is described.

3. Similar ligations may prove valuable in the management of postoperative thrombosis and embolism.

4. In this instance, circulatory disturbance of the extremity following external iliac ligation was negligible.

REFERENCES

1. Johnston, C. H. Combined ligation and injection treatment of the varicosed great saphenous vein. *J. A. M. A.*, 109: 1359 (Oct. 23) 1937.
2. Bancroft, F. W., Stanley-Brown, M., and Quick, A. Postoperative thrombosis and embolism. *Am. J. Surg.*, 28: 648 (June) 1935.
   Bancroft, F. W., Stanley-Brown, M., and Chargaff, E. Postoperative thrombosis and embolism. *Ann. Surg.*, 106: 868 (Nov.) 1937.
3. Barnes, A. L. Pulmonary embolism. *J. A. M. A.*, 109: 1347 (Oct. 23) 1937.
4. McCastor, J. T. N., and McCastor, M. C. Reaction of sodium morrhuate injections. *J. A. M. A.*, 109: 1799 (Nov. 27) 1937.

The follicle-stimulating hormone often excreted in human urine after spaying or castration or after the menopause probably originates in the anterior pituitary.

From—"The Physiology and Pharmacology of the Pituitary Body," vol. 2, by H. B. Van Dyke (University of Chicago Press).

EPITHELIAL INLAY FOR SCARS IN THE NECK DRAGGING THE LOWER JAW DOWNWARDS

J. F. S. ESSER, M.D.

MONTE CARLO, MONACO

MISS E. S. O. fell into the fire during an epileptic fit on May 6, 1934, burning her entire face and the front of her neck. She had previously been a beautiful Spanish girl. (Fig. 1.) We realize by comparing this photograph with those taken after the accident the enormous difference in the girl's appearance and how essential it is that there should be a special institute for such seriously mutilated cases.

Figures 2 and 3 show the result of an attempt made by a general surgeon to heal the ectropion of the left eye. The two flaps which were used were taken from the right part of the forehead and from the left cheek respectively, causing grave disfigurement. The right eyebrow was lifted almost an inch above the left one. The most serious disfigurement was the strong vertical deep scar under the eye, caused by the sewing of the flaps.

I will discuss here only the treatment of the neck, and not of the three epithelial inlays which formed two new lower eyelids and a new lower lip. The platisma myoides, i.e., all the directly underlying muscles of the front part of the neck were completely scarred, but the skin did not show general mutilation.

FIG. 1. The patient before mutilation.

FIG. 2. Result of an attempt made by a general surgeon to heal the ectropion of the left eye.

In Professor Estella's University Clinic, Madrid, the corpuscula carotideo was cut away, January 18, 1935, for the treatment of the epilepsy, but in spite of the greatest care, both incisions left very disfiguring scars. The vertical folds, seen in Figures 2 and 3, show clearly that the patient could not lift her chin at all, neither could she turn her head. It was necessary for her to turn her whole body if she wished to look to the side.

I operated on her on March 8, 1935, making simultaneously four large epithelial inlays, all of which healed completely. An incision was made across the front part of the neck, through the skin and the superficial muscles, close to the sternum, the portion of the neck usually covered by a necklace. This avoided a visible scar near the face.

148

The edges of the cut were pulled as far apart as possible, and a large mass of "stent" (a material used by dentists for taking moulds of

FIG. 3. The same, full face.

the jaw when making dental prostheses) was placed over it. This material is softened in hot water and stiffens in a few minutes.

The mould of the wound was covered by a thin skin flap (Thiersch) taken from the thigh (inner aspect). The mould was wrapped in the skin flap, the outside towards the mould. Then the covered mould was replaced exactly in the position in which it had been taken. It was kept for a week tightly pressed on the wound by a special bandage (in this case cotton wool and four strips of leucoplast placed tightly round the neck). A week later it was removed, the

FIG. 4. Result of the epithelial inlay on the neck, three weeks after the healing, the lower eyelids being also reconstructed by epithelial inlays.

graft having completely healed. The patient was able to move her head in every direction and her lower jaw was quite mobile.

THE NEW "MOORHEAD" ELECTRIC BONE OPERATING SET

Henry H. Ritter, M.D., F.A.C.S.

Associate Professor of Clinical Surgery New York Post-Graduate Medical School and Hospital (Columbia University)

NEW YORK CITY

THE surgeon, like the mechanic, seeks simplicity and perfection in all employed mechanisms. Cumbersome and

FIG. 1. Cutting an inlay graft with the twin saws, with the guard in place.

intricate tools are discarded when their limitations become apparent and when new simple and superior apparatus is available. The new Moorhead electric bone operating set is therefore of interest in bone surgery. Its advantages are:

1. It can be used in every type of bone surgery where bone grafting, bone drilling, bone notching, or the insertion of wires is indicated.

2. The motor can be used on direct or alternating current and can be attached to any electric light outlet.

FIG. 2. Small single saw cutting lower end of graft.

3. All parts except the motor can be sterilized. Since the motor is at a distance from the operative field, it does not require sterilization.

4. It is compact and portable, weighing 22½ pounds, including the case.

5. It consists of three main parts, the motor, the cable, and the chuck or handpiece. Special tools fit readily into the chuck.
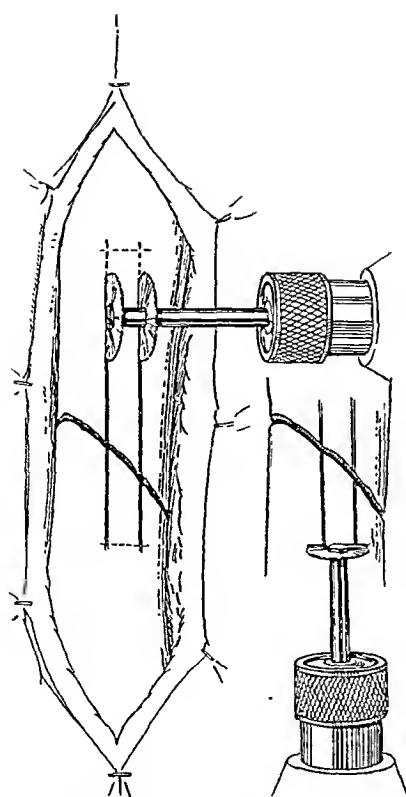
Fig. 3. Drawing of the twin and single saws cutting grafts.

CREST OF TIBIA
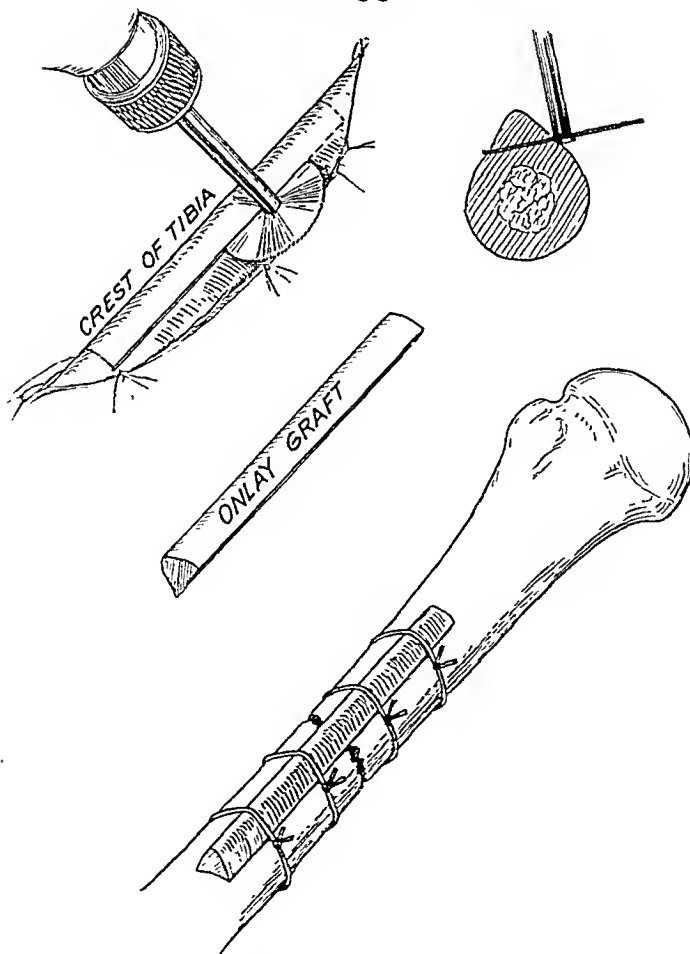
ONLAY GRAFT

Fig. 4. Cutting an onlay graft from the crest of the tibia for use in fracture of the humerus.

6. It requires very little attention. Vaseline on the inner cable and separation and cleaning of the major component parts after usage are all that is necessary.
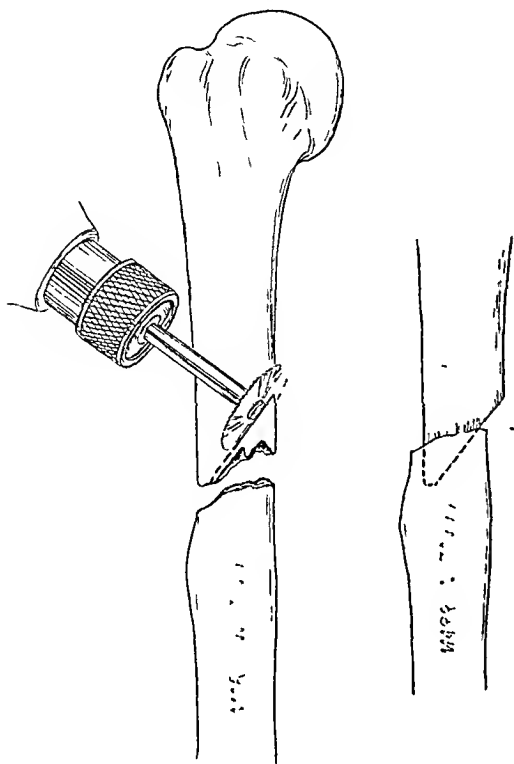
FIG. 5. Steepling the upper end of the humerus for use in open correction of humeral fracture.

stop the motor at the operator's will. The motor, being at a distance from the operating table does not get wet, thereby preventing the possibility of short circuiting.

The foot switch is attached to the motor by a length of wire so that it can be placed in the most satisfactory and comfortable position for the operator. The pressure of the operator's foot starts the motor and increased pressure accelerates it.

Cable. The cable is 5 feet long and can be made longer or shorter. We have found the 5 foot length most satisfactory. The cable itself is similar to that used on the dental motor and automobile speedometer. It is flexible and permits use of the apparatus at any angle. It consists of two parts, an inner metal cable and an outer rubberized cover. A slot at the distal end of the metal cable fits directly into a pin on the motor, and the outer cable fits over it. The latter also has a slot which is fixed to the motor by a set screw. The proximal end of the inner cable fits into the chuck or handpiece and the outer cable is screwed to the chuck.

The Chuck or Handpiece. The chuck is tubular in construction and weighs $2\frac{3}{8}$ pounds. It fits easily into the palm of the hand, facilitating one-hand control. It is so

FIG. 6. Sending drill holes through bone for passage of sutures.

Motor. At full speed, the motor generates $\frac{1}{8}$ horsepower. It is controlled by a foot switch which can start, accelerate, and

constructed that it cannot obstruct the operator's view, and with the flexible cable, it permits operation at any angle. The

various attachments fit accurately into the handpiece and do not slip. To insert any attachment it is necessary to pull down on

The attachments consist of parallel or twin saws, single saws, saw guard, drills, burrs, wrenches, a small chuck and a

SLIDE-GRAFT REMOVED FROM GROOVE
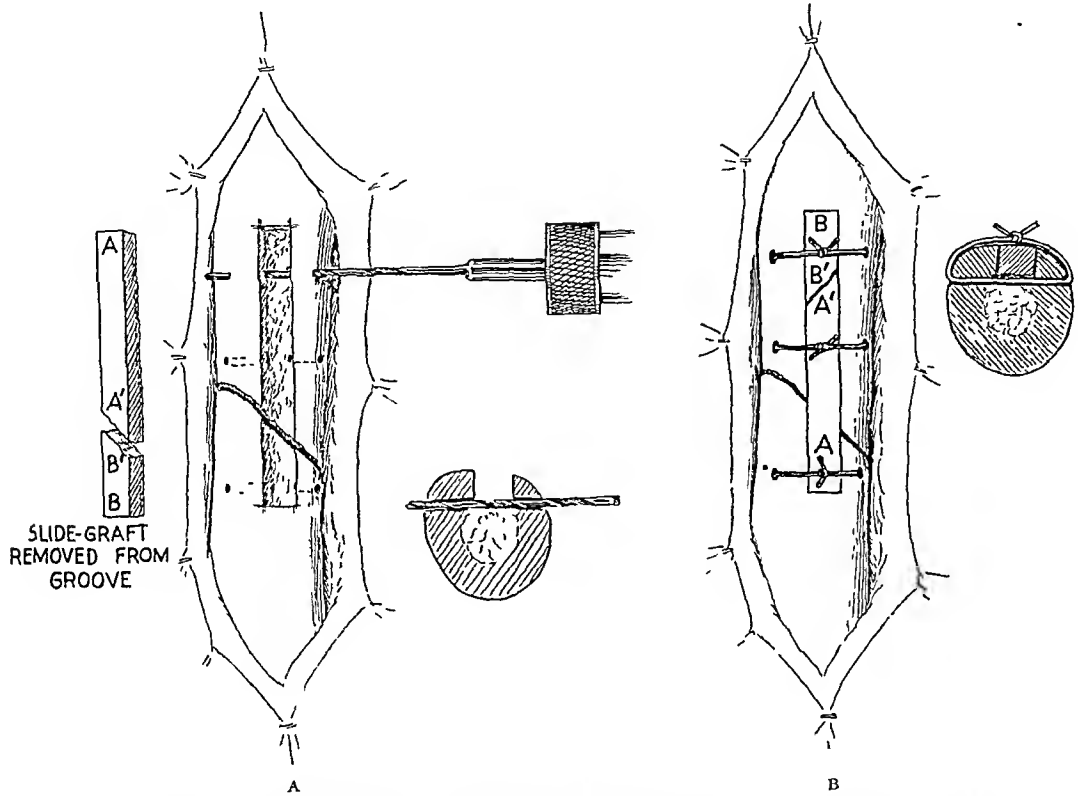
Fig. 7. A, schematic drawing of hole drilling through bone. B, graft in place and sutures tied.

SMALL SKIN INCISIONS

SKIN SURFACE

FRACTURED   BONE
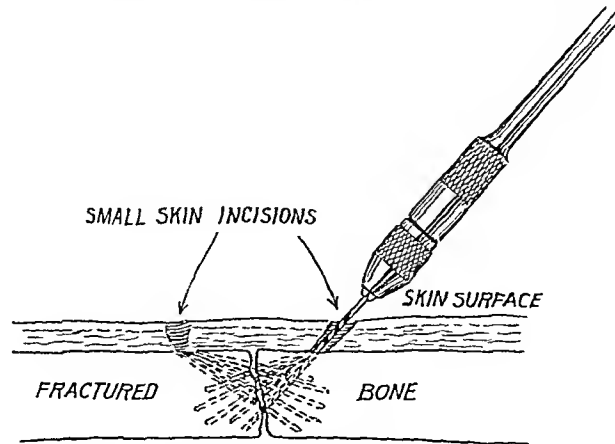
Fig. 8. Multiple drill holes for nonunion or delayed union of bone.

the purposely roughened portion of the chuck, drop the stem of the drill or saw into place, release the pull, and draw up gently on the drill or saw. The part is then firmly and securely fixed and can be released by reversing the procedure.

Kirschner wire introducer. All the saws are made of stainless steel and are circular. Each of the twin saws and the large saw are 1¾ inches in diameter and the cross-cut or small saw ¾ inch in diameter. The thickness of each saw is .045 inches. The twin

saws are made so that the distance between them can be regulated for cutting a graft of any desired width, an essential for sliding
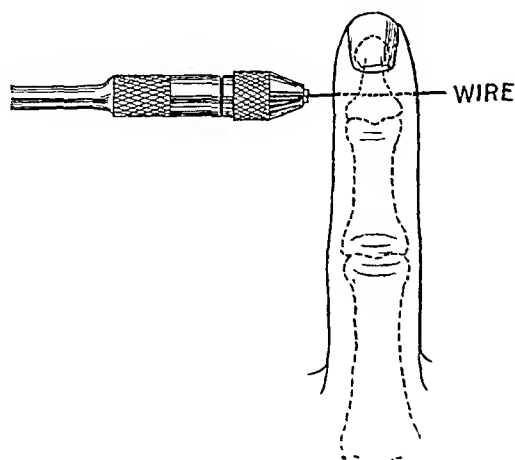
FIG. 9. Passing of wire through terminal phalanx with use of small chuck.

and inlay grafts. Curved grafts can also be made. The small single or cross-cut saw is used to cut across the upper and lower ends of the graft or for the preparation of small

Stainless steel drills of several sizes are used for making holes for the passage of sutures, for multiple drill hole treatment in non-union of fractures, or wherever drill holes are indicated. In this way a small hole may be made in the bone prior to the insertion of a pin or nail in the open treatment of fractures of the neck of the femur. The drills are excellent for making holes in the outer table of the skull where a loss of soft structures covering the skull makes granulations necessary prior to skin grafting. They are ideal for drilling a bone abscess, for diagnosis, treatment, or both.

When small drills are needed, a small chuck, fitting into the handpiece, is used. This small chuck will take and hold the finest drill. It is particularly valuable when drilling small bones such as the phalanges or metacarpals.

The Kirschner wire inserter, formerly an extra part of the apparatus, may now be directly attached to the handpiece. A set screw is fixed to a small dent on the chuck.
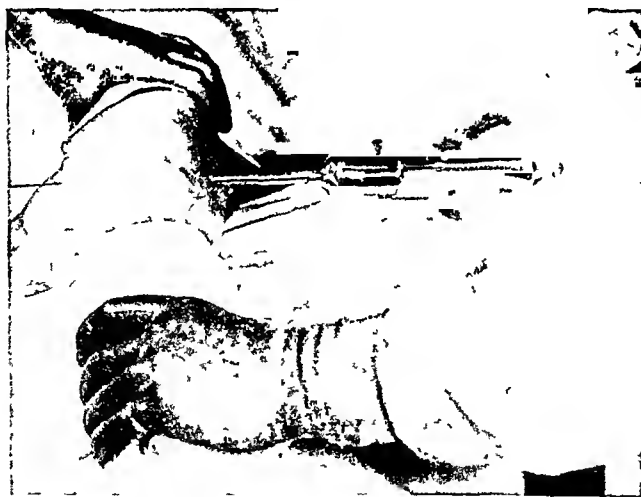
FIG. 10. Kirschner wire passed through lower end of femur.

chip grafts. The large single saw cuts bone of greater thickness, readily lending itself to amputations. It is excellent for the cutting of an onlay graft and of invaluable aid in the steepling or notching of bones.

When a saw is used, a guard attached to the chuck and fitting over the saw prevents spraying of bone dust and blood over the operative field and those near it.

Aside from the many usages to which this apparatus can be put in bone surgery, it may be of service to the otologist in mastoid surgery. In autopsies, in opening the skull, in the removal of the petrous portion of the temporal bone, and wherever bone removal without damage is indicated, the pathologist will also find the Moorhead electric bone operating set a valuable aid.

SIMPLE METHOD FOR DETERMINING THE ANGLE OF INCLINATION OF THE NECK OF THE FEMUR

E. Hugh Doggett, M.D.

Orthopedic Surgeon, Muhlenberg Hospital

PLAINFIELD, NEW JERSEY

THE treatment of fractures of the neck of the femur is one of the most frequently discussed subjects in medical literature today. The present trend of treatment is internal fixation, of which there are no less than five different techniques, namely: the Albee autogenous bone peg; the Smith-Peterson nail; the Henderson lag screw; the self-released lockbolt of Cleary and Morrison; and the wires designed by Telson or by Moore.

It is not the purpose of this paper to discuss the merits of any of these methods, but to describe a simple technique for determining the angle of inclination of the femoral neck. The success of all these

FIG. 1. Silver nitrate marks on thigh and abdomen.

FIG. 2. Anteroposterior roentgenogram of upper end of femur after reduction. Lead plates indicate the silver nitrate marks.

methods depends to a great extent on the proper location of the fixation agent. Since the normal variation of the angle of inclination of the femoral neck varies from 160 degrees in children to 125 degrees in adults, it is necessary for the surgeon to determine, by some means, the direction of the axes of the femoral neck before attempting to pass through it a drill, wire or screw.

Several different methods of determining this angle of inclination have been described. The following method, designed by the author in 1935, has been used success-

fully for two years by the orthopedic staff of Muhlenberg Hospital.

Technique. After x-ray has demon-

FIG. 3. Anteroposterior roentgenogram of upper end of femur with two Tilson pins in place

strated fracture of the femoral neck, the greater trochanter on is located by palpation. The skin overlying the greater trochanter is touched with a 50 per cent silver nitrate solution or a moist silver stick. Other marks are placed distal to this, each about 1 inch beyond the preceding one (Fig. 1A), there being five or six in all. The point where the femoral artery passes beneath Poupart's ligament is located and marked with silver nitrate. Proximal to this, four or five additional dots are placed about 1 inch apart. (Fig. 1B.) The author uses silver nitrate to stain the skin because it will last several days and is not removed by either iodine or alcohol.

After the fracture has been manipulated for reduction and is ready for x-ray examination, small pieces of lead are placed on the marks on the abdomen and fastened with adhesive tape on the thigh. The pieces of

lead on the thigh should be fairly thick, at least ¼ inch in diameter, as otherwise they will fail to cast a shadow. With the lead

FIG. 4. Lateral roentgenogram in same case.

plates in position, an anteroposterior x-ray of the femoral neck is made. The lead plates are removed and a lateral view is taken.

If reduction is satisfactory, the wire is placed as follows: The anteroposterior x-ray reveals which marks in the A and B series are in line with the axis of the femoral neck. (Fig. 2.) In this instance A3 and B3 are in the desired line. The assistant holds the foot in extreme internal rotation. The surgeon inserts the wire through the skin at A3 and points it in the direction of B3, holding the drill parallel with the operating table. The wire is forced into the neck for the desired depth. Figures 3 and 4 are anteroposterior and lateral x-ray views of the wires in place.

If the surgeon desires to insert the drill or wire into the femoral neck through a lateral incision, this technique may be used with equally satisfactory results.

BOOKSHELF BROWSING

CONSTANTINUS AFRICANUS

INCUNABULA MEDICA—III

FELIX CUNHA, M.D.

SAN FRANCISCO, CALIFORNIA

IN the year 1018 there arrived at the famous Italian medical center of Monte Cassino, maintained by the Order of the Benedictin Monks, a plodding, travel-wearied individual who had passed through many lands to arrive at this destination. At the time Monte Cassino was at the very peak of its fame as a great medical center and school of learning for physicians.

This individual bore the name of Constantinus, and because he came from Carthage in Africa he was called Constantinus Africanus or Constantin the African, according to the prevalent custom of calling a man after the place from which he came.

Historians and students of medical history have apparently passed him by, and although it is recorded that he wrote and translated much, very little material is available from which his career can be studied. This in spite of the fact that contemporaries and historians for a few centuries after his time chose to place him on an equal basis with Hippocrates, Galen, and Avicenna.

His early education had been obtained in a mosque in Cairo. Therefore it is not to be wondered at that his contacts and viewpoints were essentially Arabian and Egyptian. His practical education, or rather that education derived from actual experience, had been obtained by wandering in a never-ceasing journey through many lands, always searching out new methods, new drugs, new theory.

Arriving in Italy, he first sought the school of medicine at Salerno, at the time enjoying its greatest fame, mainly because of the intellectual capabilities of its faculty. Salerno was situated in the Bay of Paestium, just south of the Bay of Naples, and because of its mild, equable climate, had gained considerable fame as a health resort. Because of its geographical proximity to Naples, which was a great shipping and commercial port, a sort of crossroads of commerce between Italian, Greek, and African ports, it came under the influence of barbaric, Greek, Jewish, Latin, and Moslem teachings and customs. These influences, however, it seemed quite capably to synthesize to its own uses, and although it acted much as a great melting pot for all of them, none predominated or was in the ascendancy until the arrival of Constantin. He brought with him that which was not entirely new, but had in it immeasurable value, namely the Arabian influence and attitude.

The enormous amount of medical experience hidden away in Arabic literature, inaccessible because of difficulties with the

FIG. 1. Folio 135 from a thirteenth century manuscript, the "Viaticum Peregrinantis" of Ibn Al-Jazzar, the Arabian, translated by Constantinus Africanus into Latin. (Presented by Mrs. Leroy Crummer in memory of Dr. Crummer to the Welch Medical Library. Reproduced through the courtesy of Henry E. Sigerist, Librarian.)

language, became available to the then modern world for the first time. Because it had so much of value, it was intriguing, attention-commanding, and took hold in Salerno like wildfire. It represented the introduction into Italy for the first time of Arabian medicine. This did not occur without attendant difficulties. The spiritual as well as the material conceptions of the Italian people were decidedly anti-Moslem. The Saracens had been established in Sicily since about the beginning of the tenth century, and almost without opposition or interruption of note, had conducted series after series of forays against their northerly neighbors. These forays had been

attended by robbery, rape, and destruction, and to the Italians the mere mention of anything Moslem therefore served to
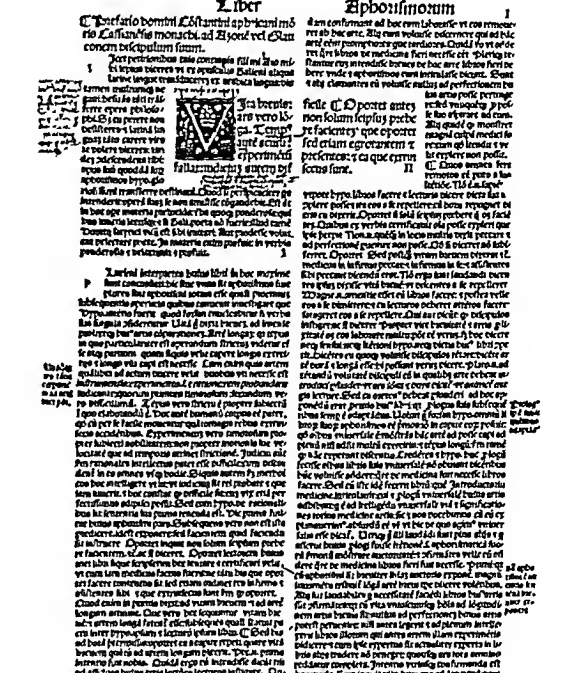
FIG. 2. Page 1 of "Liber Aphorismorum." This is the *Apborismi* of Hippocrates, with the commentary of Galenus, edited by Constantinus Africanus. (Courtesy of the Huntington Library.)

produce in most individuals a gesture of expectoration accompanied by well chosen and appropriate epithets.

Constantin went about overcoming this prejudice in a manner not without guile. In his translations of the Arabian manuscripts, instead of using the actual Arabian name of the author, he gave either its Latin or Hebrew equivalent, or attributed the work entirely to himself. After all, who was there to question this? He was the sole source and authority, and no one else had access to the works. An example of this technique was a work which he translated and attributed to Isaacus Judaeus, or Isaac the Jew. In reality this subterfuge concealed the real name of one Ishaq Ibn Suleiman Al Israili, an Arabian of Jewish origin.

Almost immediately on his arrival at Salerno Constantin was made a member of the medical faculty. In that day so great

was the fame of the school of Salerno that in any country the mere mention of the fact that one was a physician educated there, was regarded as outranking one from any other school of learning. To have been a member of the faculty was the nth degree of medical achievement.

As an individual, Constantin must have been of the inspiring type, because from the time of his admission to the faculty, the school seemed to take on a new lease of life and shook itself out of the rut into which it had fallen, to become one of the three great medical schools of Europe. This can be attributed to only one factor, namely, that Constantin began to write his Arabian translations here, and many were issued from the University in manuscript form. Gradually these manuscripts became available in other medical centers, thus increasing the fame not only of Constantin himself, but also of the school.

While at Salerno he wrote a volume entitled the "Articella," which consisted of a collection of short medical observations and translations. Most prominent among the latter was a translation of Hippocrates' "Aphorisms" and Galen's famous "Ars Parvus," and a translation from the works of a little-known Greek physician, Theophilus Protospatharios, who had lived in the first part of the seventh century and written much of value as to the changes in the pulse in disease and also of the urine in disease.

In spite of the opportunities for work, study, and fame, conditions at Salerno apparently were not exactly suited to Constantin, or it is possible that he may have had a definite desire to become a member of the Benedictin Order. His stay at Salerno was rather short one and he journeyed on to Monte Cassino. Here he was, of course, accepted enthusiastically as a distinct addition to the school, to the faculty, and to the hospital, and of still more value because of the newer knowledge which he brought to them, that of the medicine of the Arabians. Was he not the actual source from which all those arose?

He at once became a full fledged monk and embarked upon a period of tremendous work. He translated into the Latin (although historians record that it was a rather barbarous and very crude Latin, full of errors), Arabian manuscripts entitled "De Chirurgia" (Of Surgery), "De Pulsibus" (Of the Pulse), "De Febribus" (Of Fevers), "De Urinis" (Of Urine). Because of prejudices and antipathies mentioned above, he attributed most of the material in these to himself. A rather odd subject for a religious monk to write about was contained in a manuscript entitled "De Coitu."

Although the term "variola" as applied to smallpox had been used prior to his time, he brought it into popular usage by constant repetition of it in his own lectures and conversation.

Up to Constantinus' advent into Italy, almost nothing but the writings of the great Greek physicians had been studied. From his time on, introduction to the works of the great Avicenna, Rhazes, Avenzoar, and many lesser personalities—the great Arab physicians, became a part of the curriculum.

It is significant that lectures for students were available in four different languages at Monte Cassino: Jewish, Roman, Arabian, and Greek. Each member of the faculty lectured in the tongue most familiar to him. Translations were made for those who needed them by students familiar with each, who thus eked out their expenses for their own studies, very similar to the way in which modern students work their way through medical school. Constantin was familiar with all of these languages, therefore lectured and translated from any or all, as the occasion warranted.

The School itself was liberal in its views and teachings, as exemplified by its tolerance of Jews. Such tolerance did not exist in universities elsewhere at the time. It also admitted women as students and candidates for the degree of doctor of medicine. The ceremony of graduation from school and the requirements to be fulfilled before

attaining the medical degree are much too interesting to pass by, inasmuch as they show so little actual deviation from the practices of our own day, almost a thousand years later. The requirements were as follows:

First, the candidate must have attained twenty-one years of age. Second, he or she must be of legitimate birth. Third, evidence of having spent a minimum of seven years in the study of medicine must be presented. Fourth, one must have passed successfully an oral and a written examination in the writings of Galen, Hippocrates, and the Arabians, Avenzoar, Avicenna, Rhazes. These latter are of particular significance because their presence in the curriculum could never have occurred but for Constantin.

Having complied with the above requirements, the candidate promised publicly that he would administer no poisons to any one and would always treat the poor gratuitously. Having done this, he was presented with a gold ring bearing the seal of the University, a wreath of laurel, and finally, the benediction of the Holy Abbot. He was then free to go forth and practice medicine wherever he might wish.

Similar to our own day, if the opportunity were open, he could join the faculty as an instructor, for which he received a salary of 12 ounces of gold annually. At our present day price of gold, thirty-five dollars an ounce, it represented an annual income of 420 dollars, but it is not mentioned whether he received food and lodging in addition at the Monastery.

The school stressed the art and psychology of medicine rather than pure anatomy, as was the custom in the other contemporary medical schools. Symptoms, diet, and materia medica were the more important subjects. The practical approach to the patient, considering him as a human being and not as a case of something-or-other was taught.

There was some guile in these lessons, however. The student was taught always to tell the patient that he would recover, but the relatives were to be told that he was grievously ill; then if the patient died it would be said that the doctor foresaw the result and was therefore learned; if he chanced to recover, then his reputation increased that much because his skill was so great. Not poor psychology for a thousand years ago. Almost exactly the same type of guile is practiced by many today.

It is possible that Constantin may have been brushing with such a modern science as endocrinology when in describing certain conditions relating to the brain he states, "There is a hot brain which gives rise to an unstable, irascible, busy, restless individual [hyperthyroid?], and a cold brain which is the opposite of this, whose possessor is slow, sluggish, forgetful, and very sleepful [hypothyroid?]."

Epilepsy he called the "falling sickness," because "as a result of this disease, men are compelled to fall. The fall is sudden, the mouth is set awry and aside, also the face. There is a tremor of the head and of the body with grinding and gnashing of the teeth, and tight clenching of the teeth with foaming of the mouth and the pouring out of superfluities.

"They that suffer and are possessed of this evil know before hand when the evil is coming upon them [aura], for they feel the motion of some kind of breeze which comes up from the extremities. These cases are most obstinate and difficult to heal, but diet helps them." If he could only have known of "ketogenesis." Detailed study of his dietary rules might reveal him as first fostering this, with no idea as to why.

As to the therapy of epilepsy: "They must eat lightly and be temperate as to amount, taking their principal meal in the morning and little or nothing in the evening. They must be purged regularly with medicine." The roasted liver of an ass or of a goat was recommended, and, of course, leeching. But how much could we add, or improve upon that description of epilepsy as a disease?

Constantin's work was of sufficient caliber to attract the attention of the Duke

of Salerno, who invited him to become his personal physician. A few years later he was invited to return and become Professor of Medicine at the University of Salerno, a position he occupied for ten years, leaving it then only because he wanted leisure for writing, in order to tabulate the knowledge which he had gathered in his travels and in his practice.

A friend and faculty associate at the University of Salerno urged him to enter the Benedictin Monastery as a recluse, to devote himself to writing alone, and to abandon teaching and practice. This same friend a few years later became the great Pope of the eleventh century, Victor II. Each subsequent work of Constantinus was dedicated to him and sent to the Vatican Library at Rome. These manuscripts are still a proud possession of that great library.

Constantin represented one of the great intellectual minds of his time and his works were well worthy of subsequent quotation as an authoritative source of medicine. He gave a fairly good description of liver and gall-bladder dysfunction. Jaundice he described as follows: "There is dryness of the tongue accompanied by thirst and loss of sleep due to itching of the skin. The face and eyes and the skin of the whole body take on a yellowish tinge and there is a bitter taste in the mouth. The ache is on the right side of the abdomen and there is an intermittent fever."

His description of migraine, interesting to say the least, was as follows: "This ache and evil is most grievous, for whoever suffers in that way feels in his head as it were a beating of hammers and cannot bear noise nor the slightest disturbance, neither any light nor shining. This affection is caused by fumes from the stomach."

Did Constantin just barely miss giving us the first mention of intestinal allergy and its relationship to migraine? As to treatment, he advised much counterirritation, blistering.

To come down to our own day of modern advertising in magazines and billboards as displayed everywhere, he first described "athlete's foot" 700 years ago, as follows: "This disease breeds passing great itching with much rubbing and clawing on the part of the patient. As a result of this itching, many scales fall. Once this evil and malady groweth old, only with difficulty may it be healed. It hath a passion for the spaces between the fingers and the toes."

In contrast to homeopathies "like cures like," Constantin enunciated, "we heal contraries with contraries."

He instructed his pupils that unless the gravity of the illness prevented, they were always to obtain a complete history of the case from the patient himself, and never to place much confidence in the statements of wife, daughter, relatives or servants, as they might have personal aims to serve, not always honest.

With reference to the conduct of practice, he wrote, "If invited by a patient to partake of a meal in his house he should decline politely, as such familiarity soon bids a loss of confidence."

Johannes Affiacius, one of his pupils who was later to acquire great fame, wrote two treatises in medicine in collaboration with his teacher. One was entitled "De Febribus" and the other "Cura Affiacii." Both these manuscripts were later printed in book form and have survived to the present time, but present nothing in their content not found in Constantin's own works.

From the standpoint of medical bibliography, there is much interest as to what is actually available today of his writings, from which a more accurate biographical presentation might be made. Manuscripts of his, listed as being the property of certain museums, are described by their curators as being missing or lost, or, possibly, stolen. Confusion results and little is found; doubts arise and grow.

A collected edition of all Constantin's work was published in Basle in 1536. He translated the works of a famous tenth century Jewish Arabian physician, Isaac Judaeus, physician to the Caliph at Tunis.

At the personal request of the Caliph, Judaeus wrote many medical treatises, all based entirely upon his own personal observations and practice. These were written in both Hebrew and Latin, and Constantin's versatility in these languages is amply exemplified since he translated not only the Arabian pamphlets but the Hebrew as well.

If additional evidence of his familiarity with languages were desired, it was he who also translated from the Persian the work of a famous Persian physician, Haly Abbas, considered one of the three great contemporary minds in the then known world.

*This work was in the form of an en-*cyclopedia of medicine, consisting of ten separate chapters or books as they were then called, each dealing with the theory of medicine. Then followed ten chapters dealing with the practice of medicine, or more specifically, with materia medica. Included in the latter chapters was an elaborate system of diets arranged much in the manner of our own times, one particularized for each disease.

That this Haly Abbas was indeed possessed of a brilliant mind is evidenced by his attempt at a description of the circulatory system some 600 years prior to Harvey. Although lacking some of the salient points of Harvey's description, this is so close as to be uncomfortable evidence in a discussion of Harvey's claimed priority of discovery. Particularly does the description of Haly Abbas pay attention to the capillary system of blood vessels.

His keenness of clinical perception is attested to by the fact that he first advanced the idea that the uterine muscle was possessed of the power of contraction, and that birth took place by means of a succession of such contractions, his words being, "the child does not come out, it is pushed out."

Constantinus wrote his own translation of this great work under the title "Pantegni" and it was later published in printed form in Basle in 1539, as a part of a collected edition of his works.

A remarkable description of smallpox and of measles, also of coryza of head colds, to which little could be added today except as to vaccination in smallpox, was contained in another work which Constantinus translated from the Arabic, written by a very famous physician of Tunis of the tenth century, Ibn Al Jazzar, or, as translated into Latin by him, Algazirah.

There has been much bibliographical *discussion as to the authentic authorship of* a great botanical work, "Macer Floridus De Virtutibus Herbarum," which appeared about the end of the eleventh century. It was written in the form of a poem in 2,269 hexameters, describing the virtues and uses of seventy-seven different herbs and roots. Although some authorities believe it was written by a Frenchman, many concede that it shows traces of Constantinus' authorship, and may possibly have been written by one of his pupils under his own direct supervision. It represents one of the first botanic manuscripts of the Western hemisphere, is extremely rare and exceedingly valuable.

The great debt of medicine to Constantin the African, lies entirely in his having made available not only to his own but to future generations, that which might so easily have been obscured or entirely lost to posterity, namely, the remarkable surgery, materia medica, and botany of the Arabians, the Persians, and the Orientals. Of what value that contribution has been to the art of medicine let each one conjecture in his own mind.

He remained about thirty years in Monte Cassino and died there in the year 1087 A.D.

A TEXTBOOK OF NEURO-RADIOLOGY. By Cecil P. G. Wakeley, D.S.C., F.R.C.S., F.R.S.E., F.A.C.S. and Alexander Orley, M.D., D.M.R.E. Baltimore, 1939. William Wood & Co. Price $8.00.

The use of the examination by x-ray is of increasing importance in improving the accuracy of diagnosis in diseases affecting the brain and spinal cord. This new work summarizes the available data in this field.

The book contains no new material and is frankly an attempt to correlate the essential points contained in a large number of published papers and monographs on the different aspects of neuroradiology, already accepted as factual and used in many clinics.

An extraordinary mass of boiled down information is presented in these three hundred pages. Detailed application of x-ray examinations to regional areas of the skull, both in normal and abnormal cases; further discussion in disease processes; and finally its use in detailed ventriculography, encephalography, cerebral arteriography and myelography are satisfactorily elaborated, in addition to the precise method of taking films, their interpretation and the newer techniques.

Numerous illustrations add to the value of the text presentation. The material is well arranged in textbook style, with large outstanding paragraph headings, in outline form.

SIR THOMAS RODDICK. His Work in Medicine and Public Life. By H. E. MacDermott, M.D. New York, 1938. Macmillan. Price $2.00.

Anyone interested in medical history and biographies of eminent physicians will welcome this small but intensely interesting and well written volume. Especially interesting will it be to members of the profession in Canada, inasmuch as Sir Thomas was a student at McGill, a surgeon to the Montreal General Hospital, where he introduced Lister's antiseptic methods, a teacher in later years at his Alma Mater, an administrator in the medical services during the Riel Rebellion, the legislator who introduced the famous Roddick Medical Bill, and a gentleman who held a commanding position in the social and public life of his time.

It is a relaxing book to open and read of an evening. When one has finished, his wish will be that it was twice as long. The illustrations are clear and interesting. All in all, a splendid addition to Canadian medical history.

CRYSTALLINE ENZYMES, THE CHEMISTRY OF PEPSIN, TRYPSIN and BACTERIOPHAGE. By John H. Northrop. New York, 1939. Columbia University Press. Price $3.00.

This is the twelfth volume of the Columbia Biological Series. It contains the results of a series of investigations on the isolation and chemistry of bacteriophage of the proteolytic enzymes, carried out in the laboratory of John H. Northrop at the Rockefeller Institute for Medical Research at Princeton.

The general chemistry of enzymes is discussed and then pepsin, pepsinogen, chymotrypsinogen and chymotrypsin, trypsinogen, trypsin and trypsin-inhibitor, carboxypeptidase and bacteriophage are discussed in detail. The preparation and crystallization of the enzymes and a survey of the literature on the subject complete the book.

To the chemist this work may well be the last word, leaving nothing to be desired.

WHENCE? WHITHER? WHY? A New Philosophy Based on the Physical Sciences. By Augusta Baskell. New York, 1939. G. P. Putnam's Sons. Price $2.50.

This most interesting book was completed before Mrs. Gaskell's death on December 15, 1937. She had intended to make revisions and to expand it to two volumes. She was unable to do this, however, and the book is therefore presented as she left it.

The author has attempted to answer certain questions which stir vaguely in all minds. At some time in his life everyone asks himself: Is there life after death? Is there a personal God? Were we put on this earth for a definite purpose? Does science discredit the belief in God and immortality? Can we believe the Bible, in the face of scientific facts?"

"Whence? Whither? Why?" is an endeavor to answer these questions. Beautifully written,

it is a serious book, recommended to the physician whose interest in these philosophical considerations requires food, and to whom a modern viewpoint in this field, based on science, may represent an important finding.

 END RESULTS IN THE TREATMENT OF GASTRIC CANCER. By Edward M. Livingston, M.D., and George T. Pack, M.D. With a Foreword by Bowman C. Crowell, M.D. New York, 1939. Paul B. Hoeber, Inc.

Any surgeon, radiologist or clinician whose work takes him into the field of gastric carcinoma will do well to study this work by Livingston and Pack. They have analyzed 14,000 gastrectomies for cancer performed during the last half century. They have taken the data, the basis of the study, and offered the reader beautifully executed tables and charts which organize the material for ready reference. To anyone interested in the subject of the larger problem of the control of cancer, the authors have furnished a wealth of information.

Dr. Crowell in his foreword states: "The American College of Surgeons for years has been emphasizing the importance of complete and accurate clinical records in hospitals, and clinical inventories through a study of end results to form a guide as to the efficacy of the professional work in the hospital. The reading of a book such as Doctors Livingston and Pack have produced furnishes ample proof of the wisdom of those who inaugurated this phase of hospital standardization and have persisted in it in spite of results that at times have been discouraging. A model study of this nature is presented in this book and it is to be hoped that the example may be profitable. . . . "

This is not a book to be read in an evening. It is a serious study on which to form a basis for one's own hospital work.

GENERAL TECHNIQUE OF
OPERATIONS FOR
GASTRIC CARCINOMA

BY

GEORGE T. PACK, M.D.

Attending Surgeon, Memorial Hospital

AND

EDWARD M. LIVINGSTON, M.D.

Associate Attending Surgeon,
Third Surgical Division, Bellevue Hospital

GENERAL TECHNIQUE OF OPERATIONS FOR GASTRIC CARCINOMA

GEORGE T. PACK, M.D.   AND   EDWARD M. LIVINGSTON, M.D.

Attending Surgeon,
Memorial Hospital

Associate Attending Surgeon,
Bellevue Hospital

NEW YORK CITY

CANCER of the stomach under present conditions, can only be cured by means of surgery. With the rarest exception, no procedure other than excision of the tumor will save the patient with this disease from death within a short period of weeks or months. Wherever partial gastrectomies are performed, however, definitive cures result. The number of such long-term survivors leaves no doubt as to the effectiveness and utility of surgical treatment. One-fourth of patients discharged from institutional care after extirpation of gastric cancers are still living at the end of five years, and 20 per cent survive for more than a decade. These end-results compare favorably with the degree of success reached in the treatment of any internal cancer. For this reason serious operative risks are fully justified in the effort to rid the patient of his cancer through excision. Strictly palliative operations should not be considered until every possibility of removing the neoplasm has been definitely eliminated. Although rates of resectability remain discouragingly low, the basic aim which underlies every operation for this disease is a wide excision of the malignant tumor and associated lymphvascular and lymphglandular tissues whenever gastrectomy is feasible.

Surgical excision is accomplished for but a minority of patients who come under observation in any clinic. One-half or more are usually found to be in unfit condition for even exploratory laparotomy at the time when first seen. And of the remainder—those subjected to surgical therapy—fully one-half reveal intraperitoneal extensions which preclude complete removal of the neoplasm. Thus, few clinics have ever reported partial gastrectomies for more than one patient in every four observed with malignant tumors of the stomach. The surgeon, intent upon making a conscientious effort to obtain a long-term definitive cure through removing the cancer, in a majority of instances has no option but to close the abdominal incision because of hopeless findings, or to rest content with the substitution of some purely palliative measure.

It is essential that the operator be thoroughly acquainted with the complete range of these optional measures if the patient is to be afforded every possible advantage. At times an internal anastomosis between stomach and small intestine is indicated: at others, the tumor-bearing segment should be short-circuited or segregated from the remainder of the gastrointestinal tract. In many instances an external stoma must be created to give access to the interior of the organ for postoperative endogastric treatments or for the purpose of prolonged artificial feeding. Additional techniques call for use of an electrosurgical unit for the desiccation or electrocoagulation of the bulk of the tumor. Not infrequently radium or radon should be applied, either interstitially or topically for palliative effect. It is also possible to make use of contact, low-voltage *x*-ray therapy (Chaoul therapy) at the time of operation through an open gastrotomy wound.

Surgical judgment is put to a severe test by the choices involved. Certain of the operations are performed in one stage; others require two stages or multiple stages. In general, the aim is three-fold: (1) to grant a maximum prolongation of life; (2) to protect the patient from unwarranted risks; and (3) to provide for increased comfort during the fragment of life that remains where the prognosis appears hopeless.

Malignant neoplasms of the stomach show marked variations as to type, extent, location, complications, gross pathologic characteristics, relations to adjacent viscera, the presence or absence of metastases, and in the distribution of these secondary cancerous deposits when they are encountered. The utmost resourcefulness and surgical skill are called for in meeting unexpected conditions in the management of all but the small, freely movable, prepyloric cancers. The surgeon will seldom find it possible to adhere strictly to a fixed and rigid course, predetermined before actual commencement of operation. Flexibility in operative technique is the keystone to success in the surgery of gastric cancer.

CHOICE OF OPERATIVE PROCEDURES

The many operations undertaken in the treatment of stomach cancers are listed in an accompanying comprehensive table (Table 1). All are included under three general headings:

A. Exploration and simple closure.
B. Partial gastrectomies and resections.
C. Palliative operations.

The present article is general in scope; the purpose is to describe a number of technical procedures and expedients of value in a variety of circumstances, and applicable in many of the separate operative procedures. The aim is to offer methods for increasing that flexibility of technique and operative latitude or adaptability so necessary in the care of cancers at this particular site. Technical steps for trans-secting the stomach and duodenum, for vascular ligations, for gastrointestinal suturing and alignment, for the management of

TABLE I

OPERATIONS FOR GASTRIC CANCER

A. Exploration and Closure

1. Peritoneoscopic exploration.
2. Exploration through minimal incision.
3. Exploration through full-length incision.

Special Incisions	Special Types of Closure
Marwedel incision	All-layer, non-absorbable sutures
Navarro incision	Bolstered sutures (rubber tubes, buttons)
Mayo incision	Silver wire (for fascia or all layers)
Transverse incision	

B. Resections and Gastrectomies

More Common	More-Rare
1. Pylorectomy	5. Fundisection
2. Partial gastrectomy	6. Cardiectomy
3. Subtotal gastrectomy	7. Annular or sleeve resection
4. Tubular resection	8. Wedge or v-resection
	9. Total gastrectomy

Techniques of Reconstruction

(a) *On the Billroth I Principle (Anastomosis to Duodenum)*
    1. Billroth I technique   (1881)
    2. Morison-Horsley technique   (1905–1926)
    3. Kocher technique   (1895)
    4. Haberer-Finney technique   (1922–1923)
    5. Schoemaker technique   (1921)

(b) *On the Billroth II Principle (Anastomosis to Jejunum)*
    1. *The side-to-side or lateral anastomosis*
        Billroth II technique   (1885)
        Mikulicz technique   (1889)
    2. *The end-to-side or terminolateral anastomosis (oralis partialis)*
        Von Eiselsberg technique   (1888)
        Hoffmeister-Finsterer technique   (1905–1913)
    3. *The end-to-side or terminolateral anastomosis (oralis totalis)*
        Krönlein-Balfour technique   (1887–1916)
        Moynihan technique   (1922)
        Reichel-Polya technique   (1907–1911)
    4. *Special and minor modifications*
        Roux technique   (1893)
        Lahey technique
        Moynihan I technique
        Finsterer technique
        Braun anastomosis
        Dubourg technique

TABLE I (*Continued*)
C. Palliative Operations

(*a*) *Surgery alone*
1. Gastrotomy
2. Gastrostomy

Mucous Tract	Fibrous Tract
Janeway	Witzel
Beck-Jianu	Marwedel
Spivak	Stamm-Kader
Carrabba	et cetera
et cetera	

3. Gastroenterostomy
   Anterior
   Posterior
4. Palliative resections
5. Jejunostomies
   Witzel
   Marwedel

(*b*) *Surgery combined with electrosurgery*
1. Gastrotomy, plus electrosurgical excision

(*c*) *Surgery combined with radium or radon implants*
1. Interstitial gold seeds
2. Operations supplemented by teleradium or *x*-ray therapy externally or endogastric balloons applied internally.

(*d*) *Surgery combined with contact x-ray therapy*
1. Chaoul low-voltage therapy with special applicators
2. Intragastric contact Roentgen therapy

special clamps, and the like, will be described to aid in dealing with the protean manifestations of gastric tumors. The indications and contraindications which govern selection among alternative measures will also be given brief consideration. The proper selection of a method often has greater practical significance than the technical details of its actual application.

A. EXPLORATION AND SIMPLE CLOSURE

Every patient with a known gastric cancer, or known to have a gastric lesion which may prove to be a cancer, is entitled to surgical exploration unless it can definitely be demonstrated from clinical evidence that the tumor is irremovable; and, in addition, unless certainty exists that no form of temporizing operation holds reasonable promise of prolonging life or of increasing comfort. There is no guide to compare, in a settlement of these all-important points of removability of the tumor and advisability of palliative surgery, with the reliability of the evidence gained from direct inspection and palpation of the tumor and its surroundings. Definitive cures have repeatedly been secured when all preoperative data indicated no likelihood of cure. The size of a palpable gastric tumor, the

duration of the symptoms, the gastroscopic
ings when unfavorable, in fact every indi
shown to err in specific instances and at times
when curability exists. The surgeon must l
to defend his patient's interests by refusing
evidence that *may not in fact be final*. It is i
such vital import that every trace of dou
replaced by complete certainty; and in mar
sible only through the aid of abdominal se
field, if in no other, exploratory laparotomy
has distinct value as a life-saving measure.

Acceptable rates of long-term cure, furth
only from centers where operability rates h
high levels. Favorable end-results must be p
wide application of surgical methods. One
maintaining high operability rates, is the fa
where attention has been focused on the tre
have reported gastric resections for 20 to 2
observed. These high rates have been co
several consecutive decades, leaving no dou
resectable tumors where these are diligentl
free use of the exploratory incision.

Another convincing fact concerning
resectable tumors is the following: Autops
who have succumbed to gastric carcinomas
to one-fifth of instances there is no visible
cancer beyond the walls of the stomach. In
outcome has been due to dehydration or s
tions at the pylorus or cardia, to sepsis, her
other purely local causes. Many of these tur
resectable to the very end of life. To obta
only at the necropsy table is unfortunate; a
aptly termed "lost opportunities for cure."
need for the more frequent and earlier use

The remaining span of life for the avera

safe. The initial incision may be small. An opening 4 to 6 cm. long is sometimes sufficient to furnish the information desired. Such an opening can always be enlarged for further exploration or for resection. Where it is immediately seen that conditions are hopeless, this small incision may be closed at once by closely-spaced, through-and-through, all-layer, non-absorbable sutures, which will allow the patient to be out of bed, in most instances, within twenty-four to forty-eight hours. It is the routine full-length incision, with extensive intraperitoneal trauma, despite all findings, that invites undue costs and brings disfavor to the measure. The extent of exploration must be carefully adjusted to fit the gains anticipated; prolonged intra-abdominal manipulation is justified only in the face of a rising horizon of hope. Courage to terminate the operation *promptly* when conditions are found to be utterly unfavorable is almost as important as is preparedness to proceed whenever there is the slightest reasonable hope of success. It is certain that if operative mortality is unreasonably high, due to the injudicious continuance of exploration against insuperable odds, fewer explorations will subsequently be advised, and future patients, many of whom will have resectable tumors, will thus be deprived of their chance of life. Surgical exploration for this disease, when properly performed, need be attended by neither serious immediate danger to life nor by marked discomfort to the pre-moribund patient who will derive no gain from the procedure.

The scope of operation is rapidly expanded whenever conditions appear favorable on inspection through the initial minimal incision. Surgical therapy now becomes as extensive and active as conditions warrant. Gastric resection with all its potentialities for cure is always the aim, but if this cannot be performed the simple exploration is turned into some form of purposeful palliative treatment. Gentle exploration unquestionably has none of the ruthlessness that is evidenced in deliberately overriding any search for excisable tumors by means of laparotomy.

Preoperative Preparation of the Patient. Each investigative incision can be considered to constitute a potential gastrectomy. Even though it is feared that the operation will be short and end in failure, the preoperative preparation of the patient should be thorough. This concept will be amply repaid by a minimum operative mortality for simple explorations, but of even greater significance, it ensures markedly improved chances for survival where the operation suddenly becomes extensive and when extirpation is attempted.

Short-cuts in preoperative routines cannot be taken with impunity and curtailments will prove costly.

Posture of the Patient and Preparation of the Field.   The patient is placed in the dorsal decubitus with head and thighs slightly elevated. This "modified jack-knife position" aids in relaxing the abdominal wall. A small sandbag beneath the lower dorsal vertebrae throws the stomach forward and increases accessibility. The skin sterilization extends from nipples to pubes. Painting this wide area allows extension of the incision to any point on the anterior abdominal wall and over the lower thoracic cage where this is found expedient (Marwedel incision: Baudet-Navarro incision). Sterile drapes are so applied that redraping is never required.

Anesthesia.   Field-block anesthesia or local infiltration, with premedication, is ideal for simple exploration. A 0.75 per cent solution of novocaine is adequate. The epigastrium is supplied by the sixth, seventh, eighth, and ninth intercostal nerves and the entire region may be blocked along the subcostal lines of infiltration illustrated in Figure 1. Thorough injection of the parietal peritoneum well beyond the wound margins ensures good muscular relaxation. A power pump for delivery of the solution has distinct advantage as a time-saver and should be used when available. The gastrohepatic omentum is infiltrated with 3 to 5 c.c. of solution. Care is taken to avoid puncture of even minute blood vessels; otherwise troublesome hematomas develop. The mesocolon and prevertebral areolar tissues are injected only when this is required by discomfort to the patient from traction on the colon or jejunum. Such supplementary anesthesia is seldom necessary. Care and gentle manipulation at the outset of the exploration, avoiding pain from undue haste before each layer is anesthetized, go far toward allaying psychic disturbances and apprehension. Resections and gastrointestinal anastomoses of the most extensive types can usually be performed to satisfaction under local and regional methods alone. Inhalation anesthesia can be added, however, at any time.

Spinal anesthesia is routinely employed by some surgeons, since it ensures complete relaxation and makes possible operations of any desired extent. One centigram of neocaine for each 15 pounds of body weight (0.10 Gm. for a 150 pound patient) injected in the second or third lumbar interspace gives admirable results. At times a 12 centigram (0.12 Gm.) dose is used. Other surgeons prefer inhalation narcosis (cyclopropane, ethylene) and the intratracheal technique which allows protection of the bronchial tree from gastric contents that might regurgitate into the esophagus when the stomach is

FIG. I.
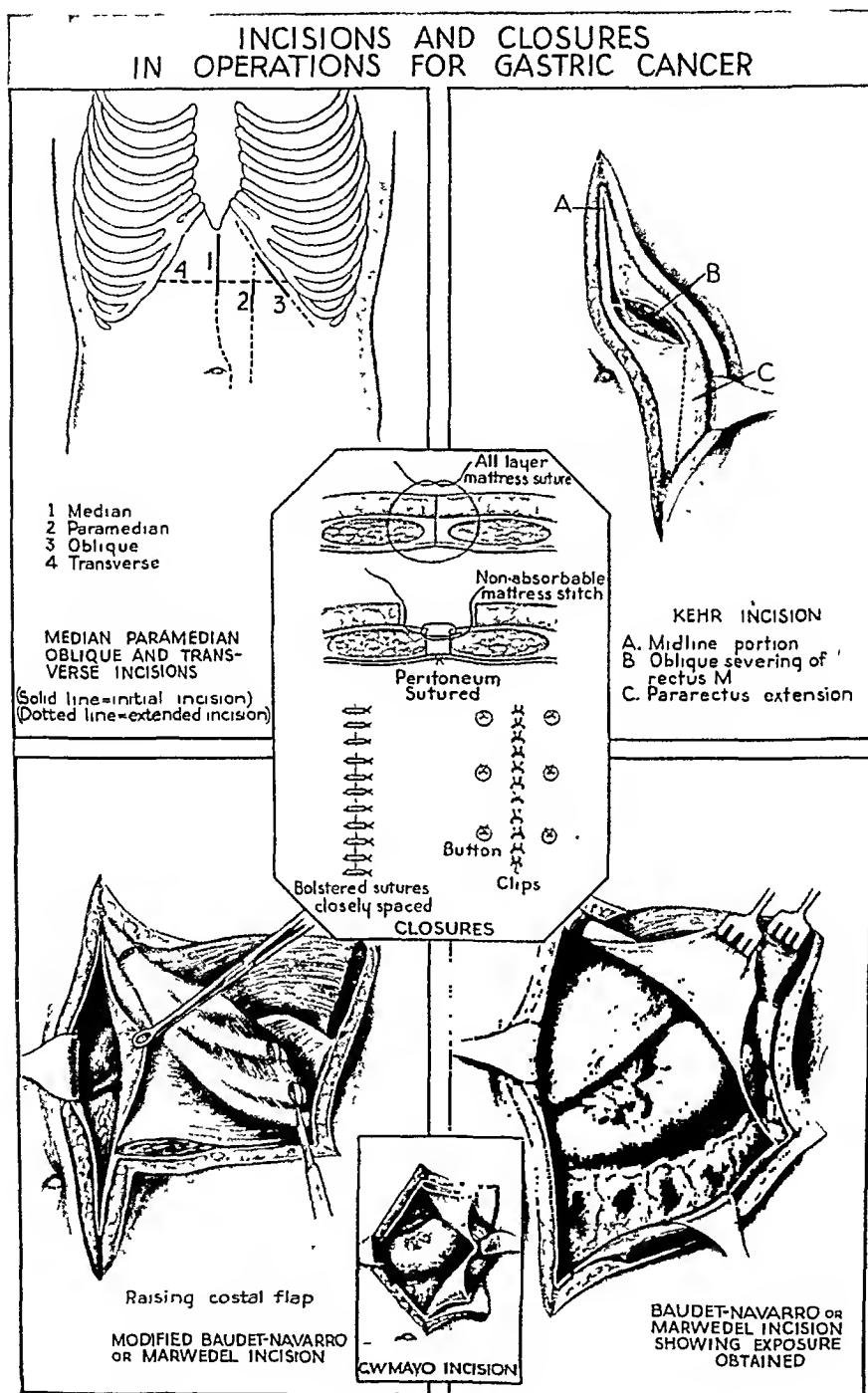
turned to the left during resection or when the stomach is pressed upon. Hyperventilation of the lungs is also possible and is advantageous when reflex muscular inhibition, pressure on the thorax, or severance of costal cartilages has depressed respirations. Each of the above methods of anesthesia has so often been employed with complete satisfaction and with such low mortality figures that the choice of technique seems largely optional and a matter of preference and training.

The Incision.   The site for the initial incision, especially when but a few centimeters in length, must be carefully chosen with due regard to the most likely site of the neoplasm as revealed by clinical, gastroscopic or roentgenologic evidence. At times this is midline, at others it lies to the left or right of the midline, and at still others, it must be placed beneath the costal margin. The wound may be perpendicular, transverse, or oblique. The incisions most frequently employed are shown in Figure 1. The dotted lines illustrate initial incisions and solid lines trace extensions therefrom.

An incision to the left of the midline avoids the falciform ligament and is preferable in most instances. The stomach is often drawn to the left by cancerous invasion of the omenta and freely movable prepyloric cancers can readily be delivered into a left upper quadrant wound or to the body surface. In addition this location facilitates anastomosis of a short gastric stump where a subtotal gastrectomy has been performed.

The Marwedel and Baudet-Navarro incisions are shown in self-explanatory illustrations. (Fig. 1.) The latter incision exposes the cardiac end of the stomach and the abdominal esophagus by creating an osteochondral flap after severing the seventh, eighth, and ninth costal cartilages with a sharp scalpel. Such an incision is rarely needed but proves invaluable where a total gastrectomy is attempted, and for radon implantation in difficult cases. Any incision may be used which provides adequate exposure; and needs in this regard vary considerably from case to case. Time should not be wasted by having too limited a field when extensive operations are undertaken; and refinements such as preserving minute nerve filaments are brushed aside in view of the lethal character of the disease.

Exploring the Peritoneal Cavity. (a) *Through the Peritoneoscope.* Every abdominal surgeon should be familiar with the diagnostic possibilities of the peritoneoscope. Hepatic metastases and peritoneal carcinomatous implants are readily visualized through this instrument. When the technical operability or inoperability of the gastric cancer due to fixity or extension into the cardia is in question, the

peritoneoscope may not give the answer. Here direct palpation is required. When the inoperability and incurability are definitely evident on peritoneoscopic examination, the necessity for even a minimal laparotomy incision is obviated.

(b) *Through a Minimal Incision.* The presence or absence of ascitic fluid is noted as the peritoneum is incised. Even small amounts indicate a grave prognosis, for this usually signifies that peritoneal invasion has already occurred. Cancer nodules on the parietal peritoneum are felt by sweeping the finger over the inner surface of the wound or are evident on inspection. The condition of the viscera of the supracolic compartment of the peritoneal cavity is also seen on inspection. A Cameron light is a useful aid. Upward traction on the abdominal wall will create negative pressure and enlarge the field of vision. The stomach itself may often be grasped by Allis clamps and gently drawn into the wound or to the body surface. Care must be taken to avoid disruption of protective adhesions or dislodgment of free fragments of cancer. As a rule the liver edge is easily exposed. Often a piece of liver or suspicious overgrowth may be excised for biopsy examination. Biopsy specimens should be secured from stomach and lymph nodes as well, when this is possible. If conditions seem hopeless the incision is promptly closed; but if not entirely hopeless, the wound is enlarged and the operation continued.

(c) *Through a Large Abdominal Incision.* Distant metastases are of greatest significance in determining unresectability, hence these are searched for first after enlarging the operative field. The pelvis and lower abdomen are explored in a search for a Krükenberg tumor or Blumer's pararectal shelf. Preoperative physical examination (pelvic and rectal examination) is not relied on in determining the condition of the lower abdomen. It is important to know the location and extent of any metastatic deposits even when a palliative resection has already been decided on. After exploring the pelvis the celiac and superior mesenteric lymph nodes are palpated. Finally the liver and the stomach itself are subjected to scrutiny. Care must be taken not to mistake a patch of hepatic cirrhosis or a benign liver lesion for a secondary implant from the stomach. The under surface of the diaphragm, the abdominal esophagus, all perigastric regional lymphatics.and each of the omenta are individually studied. The posterior wall of the stomach is reached by incising either the gastrohepatic or the gastrocolic ligaments. Occasionally the anterior stomach wall is incised for inspection of the interior of the organ.

Criteria of Inoperability; Indications for Immediate Closure. Curative resection is not possible in the presence of widespread

carcinomatous implants, of extension to adjacent viscera, or of multiple liver metastases. Involvement of the cardia, total implication of the stomach (linitis plastica type) and massive or widespread metastasis to lymph nodes are almost always contraindications to gastrectomy. The presence of a few enlarged lymph nodes along the greater or lesser curvature, however, does not always preclude cure; for in one-half of instances the enlarged nodes associated with gastric cancers prove to be inflammatory on microscopic study. A palliative gastrectomy is sometimes advisable, despite a few isolated hepatic implants since such secondary tumors are often delayed in growth after removal of the parent cancer. Radon implantation in the stomach or nodes is often advisable before wound closure. In general, surgical restraint is wise when confronted with clear-cut evidences of hopelessness. Biopsy specimens should always be secured, however, since time and again the gross pathologic picture of cancer has been simulated by lesions which proved benign on microscopic study.

Wound Closure.   In the majority of patients with gastric cancer it is wise to anticipate delayed wound healing and to make use of non-absorbable materials for suturing the abdominal wall. Heavy silk interrupted stitches are placed as close as ⅛ inch apart. All layers are similarly sutured. Fine wire may be employed when desired for repair of the fascia, relying on this layer for security. Other types of closure make use of various forms of bolsters and buttons beneath retention sutures. Drainage is seldom necessary, but is used in the presence of obvious peritoneal contamination by infectious material. It is well to make wound closure sufficiently secure to allow the patient out of bed at the earliest possible time, since this reduces pulmonic complications. Patients who do not receive gastrectomies should be ready for discharge to radiation therapy as soon as can be arranged; and those in hopeless condition should be allowed to return to their families and friends at an early date unless continued hospitalization is deliberately chosen or proves imperative.

Life Salvage from Exploration in Clinically Hopeless Cases.   It has repeatedly been emphasized that a clinical appearance of hopelessness in cases of gastric carcinoma is by no means always confirmed by laparotomy or necropsy findings. Unexpected instances of resectability are disclosed in a definite percentage of patients undergoing exploratory laparotomy. The highest rates of resectability have always been in association with the highest reported rates of operative investigation.

But the life-saving value of explorations does not rest with this consideration only. Non-cancerous conditions of the stomach are

always disclosed in an additional percentage of patients, despite the preoperative diagnosis of gastric carcinoma. Many such lesions prove to be surgically curable in character. The operative findings include pancreatic cysts, abscesses within the confines of the lesser peritoneal sac or retrogastric space, omental adhesions, cholecystic tumors and various biliary abnormalities, notably atypical forms of cirrhosis; and of even greater incidence are benign penetrating ulcers of the stomach, gastric polyps, duodenal diverticula and duodenal ulcerations with periduodenal and peripyloric complications. The authors have observed each of these, some in several instances, at operations on individuals thought to have gastric cancer.

Diagnostic errors seem infrequent on the basis of casual impressions, yet accurate records always reveal them in any hundred cases studied; and wherever necropsy percentages are high unexpected findings are disclosed the more certainly and relentlessly. Estimates as to the incidence of such diagnostic errors vary from 2 per cent to as high as 10 per cent. The fact of their infrequency in specialized institutions for the treatment of cancer does not lessen their importance elsewhere and the possibility of diagnostic error must always be borne in mind. Exploratory laparotomy for gastric carcinoma and suspected gastric carcinoma, despite any disheartening aspects, is an operation accompanied by a predictable salvage of human life far in excess of that generally accorded this important surgical procedure.

B. PARTIAL GASTRECTOMIES AND RESECTIONS

Partial gastrectomy for cancer is an operation of two distinct parts: (1) removal of the malignant tumor; and (2) restoration of gastrointestinal continuity. The object of the operation is completely fulfilled in step one, the extirpation of the cancer and associated lymph nodes. Each of the several names applied to the procedure—gastrectomy, extirpation, excision, resection—defines the operation as one for removal of the tumor-bearing segment. Step two, the repair of the parts that remain is a matter of secondary importance and represents but a necessary aftermath of the resection proper.

These divisions are sufficiently distinct in their purpose and technique to demand separate consideration; and in practice it is advantageous that they be viewed as constituting in a sense two different, but related, operations. When undue emphasis is placed on the stage of repair, or when gastrectomy is considered from beginning to end as a single unified measure, there is a tendency to modify

the excision of the cancer so that it may fit some preconceived plan for subsequent gastrointestinal anastomosis. As a consequence thoroughness is often sacrificed to convenience. A sufficient number of technical methods are now available for safely reconstructing the gastrointestinal canal to meet any situation that will be encountered after excising the most extensive gastric neoplasm. More than a score of such procedures are outlined in an accompanying table. The decision as to the specific type of anastomosis to employ in a given case should be withheld until the malignant mass has been mobilized or completely removed and the exigencies of the individual operation determined. No stereotyped operation can prove suitable for all gastrectomies. But while details concerning the restoration of the integrity of the digestive tract prove of relatively minor significance, the extent of the resection is always a matter of major importance.

<div style="text-align:center">REMOVAL OF THE MALIGNANT TUMOR</div>

The following structures must be severed in the excision of a gastric cancer:

1. The omenta.
2. The four major gastric blood vessels.
3. The duodenum.
4. Abnormal adhesions, when present.
5. The proximal segment of stomach.

Each of these structures is individually dealt with by the surgeon in orderly progression.

1. *Transsection of the Gastric Ligaments. Severing the Omenta.* Dissection begins by severing either the gastrocolic or the gastrohepatic ligament. The omental bursa or lesser peritoneal cavity is thus entered which gives access to the posterior wall of the stomach. One of two courses may be taken in the commencement of this transsection of the gastric attachments: (*a*) the surgeon may begin opposite the site of greatest cancerous involvement of the stomach; or (*b*) he may start as far as possible from the tumor. Each course has advantages and advocates. An incision opposite the site of maximum involvement allows the most difficult field to be surveyed at the outset, which means that the operator may turn back before important blood vessels have been severed if this seems advisable. But when a point far from the tumor is selected for the starting point all difficult dissecting is done only after the stomach and tumor-bearing segment have been extensively mobilized. Both advantages cannot be had and the operator must choose his course. No hard and fast rule need be followed save that of individualizing.

SEVERING THE GASTRIC ATTACHMENTS OR OMENTA

Clamp and Ligation Method
METHOD I

Aneurysm Needle Method
METHOD II

Suture-Ligature Method
METHOD III

Forceps-Ligature Method
METHOD IV

Hand within lesser peritoneal cavity or retro gastric space.

Exposure of middle colic artery. (Loop of jejunum has been drawn through transverse mesocolon)
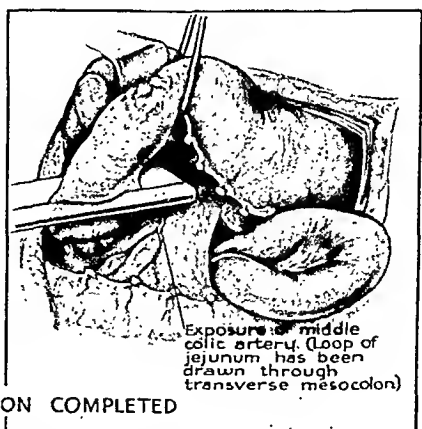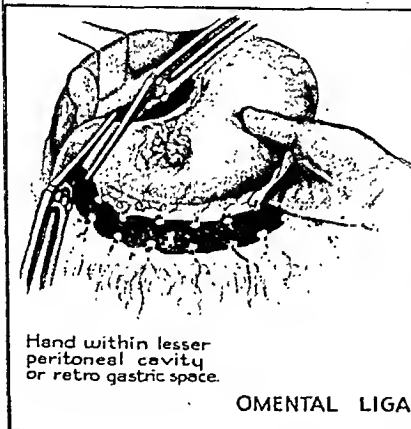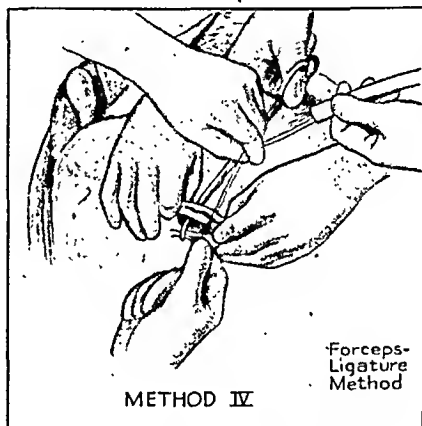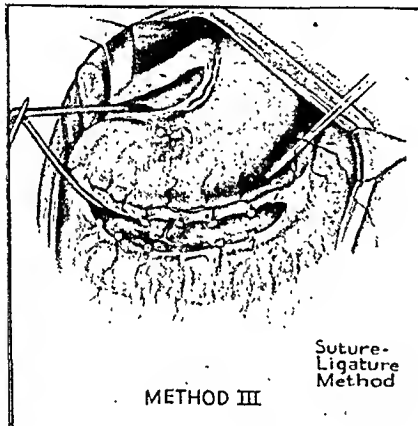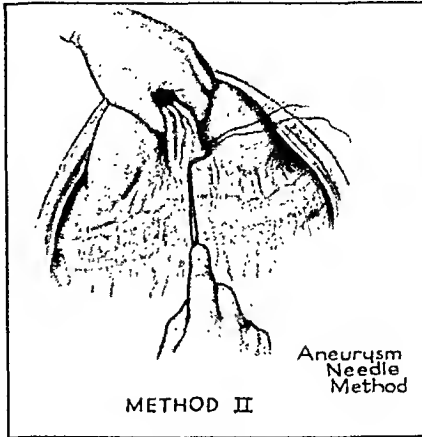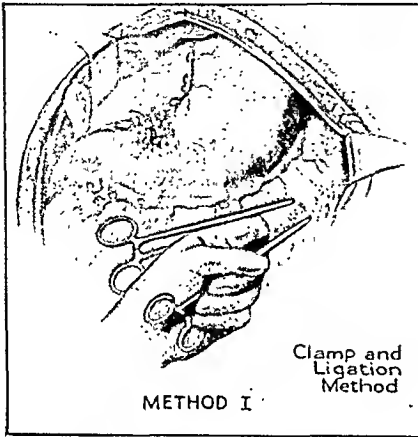
OMENTAL LIGATION COMPLETED

FIG. 2.

There will seldom be difficulty in any specific case in seeing at once which course is preferable. As a rule the first opening is best made far from the tumor at a point just below the greater curvature and well to the left of the midpoint of the stomach. (Fig. 2.)

During gastrocolic ligation the structure is rendered tense by an assistant who holds the stomach upward and the transverse colon downward. A bloodless area is selected and the retrogastric space entered by either sharp or blunt dissection. Care is taken to incise only the gastrocolic ligament itself and to do no damage to the transverse mesocolon lying beneath. The ligament is now severed, step by step, until the region of the pylorus and proximal duodenum is reached. The line of incision lies well toward the transverse colon in order to include all infragastric lymph nodes with the portion of stomach to be removed. It is well to keep the middle colic artery within the transverse mesocolon under full vision as each portion of omentum is clamped or tied. In the neighborhood of the gastric antrum the gastrocolic ligament and transverse mesocolon may be adherent, but unless carcinomatous invasion is present at this point these structures are easily separated by blunt dissection. Details concerning the arrangement of the peritoneal leaves of the gastro-colic omentum in the neighborhood of the pylorus are found under the heading *Ligation of the Gastro-Epiploic Vessels.*

The vessels of the omenta may be dealt with by one of several methods: (1) by double clamping, cutting and tying (Fig. 3); (2) by double ligation with the aid of aneurysm needles; (3) by double ligation by means of suture ligatures; (4) by carrying doubled ligatures through bloodless areas with hemostats. When needles are used these are passed eye-foremost to protect the vessels against puncture. Whatever method is chosen, the ligations are made at a distance from the stomach since the vital question of recurrence may depend upon the matter of leaving no metastatic foci behind within the subgastric lymph nodes.

Injury to the middle colic artery is the greatest danger in this stage of the operation. This is usually avoided with ease by keeping to the right line of cleavage and with the vessel under full vision at all times. When injury occurs the transverse colon is deprived of blood supply. This will often be severe enough to necessitate adding resection of a portion of the colon to the procedure—greatly prolonging the operation and diminishing the chances of success. At times, although rarely, the vessel is deliberately cut because of inseparable cancerous adhesions. When this is done, or when accidental injury occurs, any treatment of the colon is best delayed until conclusion of

the excision of the gastric cancer. At times the devascularized colon may be simply exteriorized at the end of the operation; at other times the severed ends may be delivered from the abdominal wound after colic resection, leaving a double-barrelled colostomy to be dealt with secundum artem; and at still other times the resection may be followed by immediate end-to-end anastomosis. These options are given in the order of preference. Due care in severing the gastrocolic ligament leaves little prospect of damaging this important middle colic artery.

Transsecting the Lesser Omentum.   The gastrohepatic or lesser omentum is of great tenuity and almost entirely bloodless, save at its right and left extremities. It may be bluntly torn through near the liver with impunity. While the arteries to the greater curvature are compound vessels (gastro-epiploic arteries) which supply both stomach and ligament, the arteries of the lesser curvature are not compound (the gastric arteries, right and left) and send no blood to the lesser omentum; at least they furnish no sizable vessels. Hence the gastrohepatic ligament may be torn without fear of hemorrhage. A hand introduced into the lesser sac or retrogastric space after gastrocolic omental ligation aids in making the lesser omentum prominent. If desired, a rubber tube or gauze band may be thrust through the lesser omentum to encircle the stomach as a sling and provide strong traction-control of the viscus.

The initial hole in the omentum is enlarged, first toward the cardia, then toward the pylorus. The region of the cardia, containing the left gastric artery is best dealt with later in the operation after the duodenum has been severed and the stomach has been turned to the left. Details for severing the right and left gastric arteries are discussed later. But the left gastric artery is encountered at this point and is ligated in close proximity to the stomach in a pylorectomy but near its point of origin from the celiac axis for a subtotal or total gastrectomy.

2. *Ligation of the Gastric Blood Vessels.*   It matters but little which of the four major gastric arteries is ligated first. Sites of ligation are determined by the proposed lines of transsection of stomach and duodenal walls; all ligations are made well beyond the limits of the cancer.

Ligation of *the left gastro-epiploic vessels* offers no difficulty. Suture ligatures of No. 1 plain catgut are used, with a round-pointed full-curved needle of small caliber. A small bite of stomach wall may be included to prevent slipping. Double ligation is advisable to the proximal side of the point of section. The vessel lies 1 cm. or more

below the greater curvature between the layers of the gastrocolic ligament. (Fig. 3.)

The right gastro-epiploic vessels are similarly dealt with as they course within a leaflet of peritoneum which runs between the hepatic flexure of the colon and the pylorus. Numerous small veins accompany the artery within this fold. The vessels are ligated en masse with the aid of suture ligature or aneurysm needle. A mass of omentum 1 cm. in diameter just below the pylorus is encircled and this safely ensures catching the artery; if a suture ligature is used a small bite of duodenal wall may be included to prevent slipping. The vessels are doubly or triply ligated and in the latter case are cut between the distal two ties. *The gastroduodenal artery* requires ligation only in extensive mobilizations of the duodenum; it lies well out of the way on the posterior abdominal wall and can be pushed away from the duodenum in most cases by blunt dissection. *The superior pancreaticoduodenal vessels* course beneath the posterior and inferior duodenal wall and seldom need to be disturbed; if ligation is advisable for wide duodenal mobilization a suture ligature, catching a fragment of duodenal wall, secures the artery; nutrition of the duodenal stump is ensured from the anastomosis to the inferior pancreaticoduodenal artery, the uppermost branch of the superior mesenteric. After the vessels immediately below the pylorus and duodenum have been dealt with the operator may insinuate his fingers behind the duodenum and gently separate this structure from the pancreas by blunt dissection.

The right gastric or pyloric artery is ligated within the lesser omentum as the vessel approaches the pylorus. At times the right gastric artery arises from a common trunk with the gastroduodenal artery, but this has no effect on its ligation. The gastric vessels are clamped, cut and doubly ligated as the right extremity of the gastrohepatic ligament is mobilized. The important vessels and duct within the free extremity of the lesser omentum at the foramen of Winslow lie well posteriorly and to the right of the field of operation. Ligation of the right gastric vessels is made sufficiently far from the pylorus to avoid any suprapyloric lymph nodes, which are thus removed with the neoplasm.

The only remaining major gastric vessel is the *left gastric or coronary artery*. This vessel approaches the cardia within a semilunar fold of peritoneum known as the left gastropancreatic ligament, or falx coronaria, which it traverses in passing from the posterior wall of the omental bursa (lesser peritoneal cavity) to reach the gastrohepatic ligament. For high ligation the vessel is tied within this fold

as near as convenient to the point of origin from the celiac axis. High
ligation cannot be accomplished until the duodenum has been
severed and the stomach as a whole has been turned toward the left.
This ligation, therefore, is the last to be made in dealing with the
gastric vessels. Adequate removal of the paracardial lymph nodes
and those high on the lesser curvature demands this high ligation.
When the stomach is rotated toward the left, care must be taken to
avoid spilling gastric contents into esophagus from which it might
gain access to the bronchi; here it is well to aspirate the stomach, if
it is not already empty, by making a small stab-wound well away
from any point of neoplastic invasion of the wall. The flaccid empty
organ is easier to manage during ligation of the vessel. For resections
of limited extent the coronary (left gastric) artery may sometimes
be ligated along the lesser curvature well below the cardia. This
point of low ligation lies below the cardiac branch of the artery
which supplies the abdominal esophagus and cardia. The proximal
stump of the vessel is doubly ligated, after which the omental fat
below the point of ligation is dissected downward to be removed
with the tumor, and any raw surface thus created is inverted by
interrupted sutures. When there is much fat in the lesser omentum
the gastric artery may be hard to identify. An accompanying illus-
tration (Fig. 3) shows how the thumb and forefinger of the left hand
may be used to palpate the edge of the lesser curvature and aid in the
passage of a suture ligature or aneurysm needle beneath the vessels.
A fine-pointed hemostat may also be used to create a tract beneath
the vessels, being pushed through the omentum under guidance
of the thumb and forefinger.

All gastric vessels are of rather small caliber (smaller than radial
arteries) and if accurately identified and carefully approached will
be easily ligated. Inaccurate identification may result in troublesome
hematomas between omental layers before the vessels are caught.
Immediate control of the blood supply of the stomach may be
secured at any time by placing hemostats on all important arteries
simultaneously. This expedient can be followed by leisurely deliga-
tion of the individual vessels. Yet it is always preferable to deal with
each vessel in rotation by primary suture ligatures.

A number of *small but important veins in the region of the head
of the pancreas* must be dealt with where the duodenum is widely
mobilized. These vessels encircle the duodenum, passing toward the
head of the pancreas in two vascularized strands of connective tissue,
one layer leaving the anterior and the other the posterior wall of
the duodenum. These layers are carefully identified and caught with

LIGATING BLOOD VESSELS OF THE STOMACH

Vessel 1
LIGATION OF LEFT GASTRO-EPIPLOIC ARTERY

Vessel 2
LIGATION OF RIGHT GASTRO-EPIPLOIC ARTERY

Vessel 3
LIGATION OF RIGHT GASTRIC ARTERY

Ligation of vessels within the walls of the stomach

1A Left gastro-epiploic artery-Low ligation
1B Left gastro-epiploic artery-High ligation
2 Right gastro-epiploic artery
3 Right gastric artery
4A Left gastric artery (low ligation)
4B Left gastric artery (high ligation)
5 Not tied (gastro duodenal Artery)
6 Not tied (superior pancreaticoduodenal A)

Middle colic A.

Vascular strands between duodenum and head of pancreas

(cross-section)

These minute vessels encircle the duodenum and are ligated as two strands (only necessary for wide mobilization of duodenum)

GASTRIC LYMPHATICS

Vessel 4
LOW LIGATION

THUMB AND FOREFINGER GUIDING ANEURYSM NEEDLE

HIGH LIGATION NEAR ORIGIN FROM COELIAC AXIS

LIGATION OF LEFT GASTRIC OR CORONARY ARTERY

FIG. 3.

clamps or secured by ligation with aneurysm needles. Clear visualization renders this ligation safe and readily performed.

Rarely the *splenic artery* may be damaged in resections of unusual extent. In such emergencies this vessel may be ligated with no apparently serious consequences.

The vasa brevia are only dealt with in fundisections, total or subtotal resections and are easily secured in the layers of gastro-splenic omentum. For total gastrectomies a few additional vascular twigs must be secured as they traverse the antrum cardiacum and abdominal esophagus. They lie close to the under surface of the diaphragm in a peritoneal fold known as the gastrophrenic ligament and represent anastomotic branches of the left gastric artery (cardiac branch) with the left inferior phrenic artery.

Management of the vessels within the coats of the stomach itself are dealt with under the heading of *Transsection of the Gastric Walls.*

3. *Severing the Duodenum.* Methods for transsection of the duodenum include the following technical measures: (1) the use of the de Petz suturing instrument; (2) the crushing clamp technique; (3) the purse-string or ligature technique; (4) the "open" method; (5) the "semi-open" method; (6) the soft-clamp technique; and (7) the use of the Dawson-Furniss clamp and pin. Each will be briefly described in turn. (Fig. 4.)

(1) *The Use of the de Petz Suturing Instrument.* The use of this instrument is illustrated in Figure 4. Bleeding and leakage of gastric or duodenal contents are prevented by use of this tool. The duodenum is severed between the two rows of metal clips left in place as the clamp is removed. Guy sutures may be placed to help control the duodenal stump as the clips are inverted by two rows of interrupted non-absorbable stitches. The cost of this instrument, its lack of availability in many instances and the fact that it cannot easily be applied to the duodenum in all cases greatly limit its use; and the surgeon should be prepared to employ other methods for duodenal closure.

(2) *The Crushing Clamp Technique.* Here the proximal duodenum is grasped by two crushing clamps (Payr, Carmalt) from 1.5 to 2 cm. beyond the pylorus or beyond any palpable cancer. Protective pads are placed behind the pylorus and duodenum and the latter is severed between the clamps with knife, scissors or cautery. The stomach, with its pyloric end covered by a protective rubber shield, is turned to the left and the duodenal stump immediately closed by a Parker-Kerr over-and-over lateral running mattress suture, reinforced by two rows of interrupted non-absorbable

stitches. Tabs of omentum are sutured over the stump or it is stitched against the head of the pancreas for further security. The chief objection to the crushing clamp technique is its devitalization of the end of the duodenal stump and the fact that a short stump is difficult to suture when a clamp is in situ, except after extensive mobilization. If the Billroth 1 principle of repair is to be employed, the duodenum is not closed but the clamp left in place until the cancer-bearing segment of the stomach has been resected and the first row of gastroduodenostomy suturing has been begun.

(3) *The Purse-String or Ligature Technique.* Here the proximal duodenum is crushed by two clamps which are then removed and replaced by a heavy single or double strand of suture material which is tied in the crushed portion of the bowel to continue its occlusion. A clamp of any type is now placed on the gastric side of this occluding ligature and the bowel severed. The ligated stump is further treated by an inverted purse-string suture to bury the occluding ligature, and by two rows of interrupted inverting mattress stitches of silk or linen.

A crushing clamp may be placed on the duodenum just beyond the pylorus. Then distal to this clamp a careful encircling seromuscular incision is made down to but not through the mucosa. The seromuscular layer retracts, leaving a redundant intact mucosal tube. After inserting a circular purse-string suture through the mucosa, this layer is completely severed and the stump inverted when the purse-string is tied. The seromuscular coats are then sutured by two rows of interrupted inverting sutures. (Fig. 4.)

(4) *The "Open" Technique.* This has many advocates and special advantages. Chief among the latter are accurate hemostasis, lack of devitalization of the remaining bowel, and ease of closure due to the lack of any cumbersome instruments. The procedure is executed as follows: Pads are placed behind the duodenum and pylorus; two guy sutures are placed, one on the upper and the other on the lower border of the duodenum, just distal to the proposed line of transsection; one crushing clamp is placed across the duodenum just beyond the pylorus or palpable tumor; the duodenum is severed by knife or cautery, hugging the clamp. Leakage from the open duodenal stump is confined to a small amount of frothy material which is controlled by gauze wipes. A few minute spurting vessels are immediately caught in mosquito clamps and separately ligated. The suction tip is kept ready for use but is seldom required. The stomach is turned to the left, covered by a rubber pad; and the duodenal stump is at once closed by three layers of inverting stitches
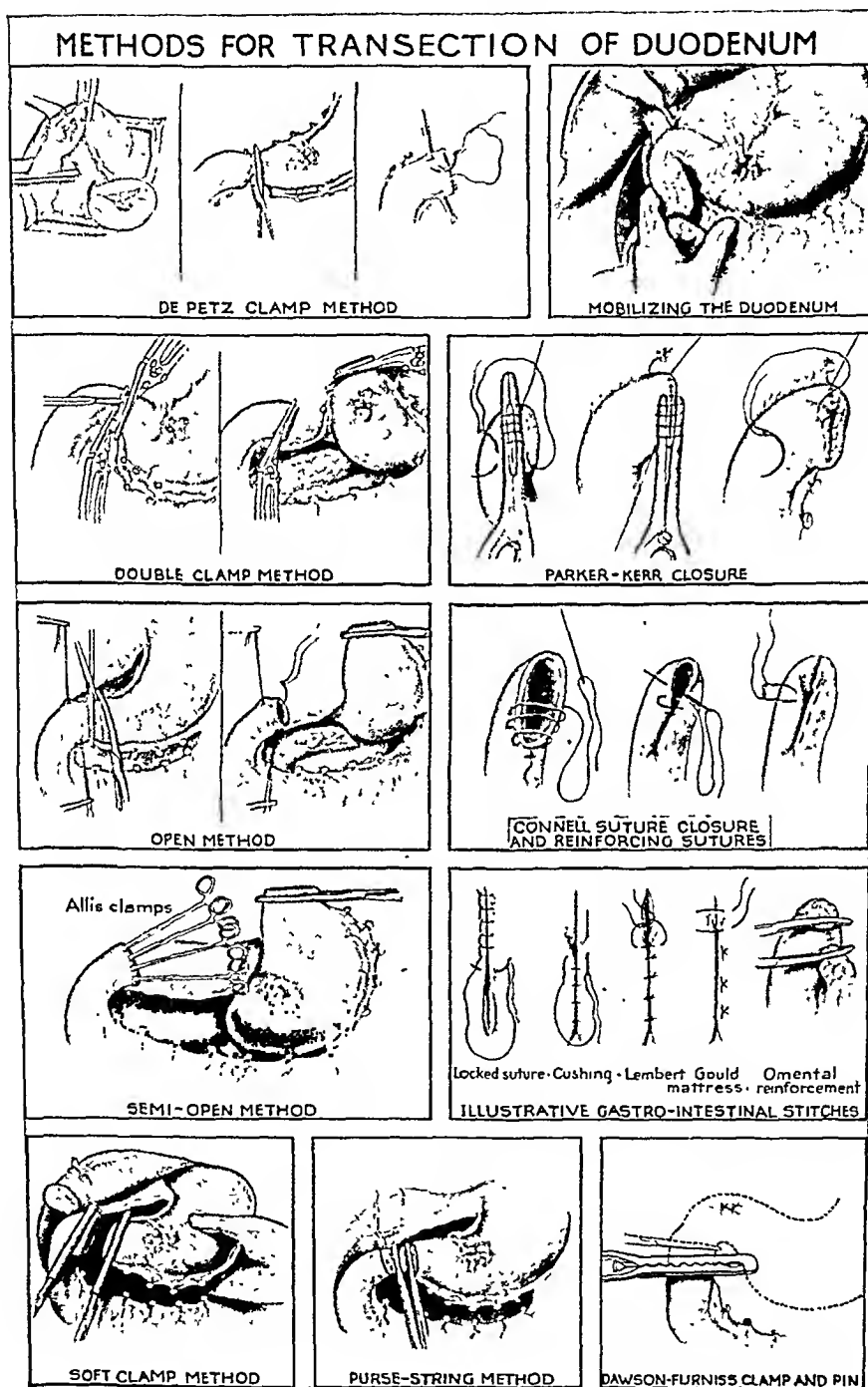
FIG. 4.

as the stump is controlled by traction on the guy sutures. The first suture row may be of the Connell variety which inverts the mucosa. This is an important aid to security, and the additional mattress sutures (Lembert, Gould, Halsted) are placed without difficulty to give wide peritoneal approximation.

The technique for passing the Connell suture is as follows: "The left hand holds the suture taut. The right hand thrusts the needle from without inward, and as the thread is partly pulled through the loop on the outside is caught by the left middle finger; at the same time the left thumb and forefinger grasp the thread at a distance from the needle. This arrangement holds the wound edge fast while the outgoing needle thrust is made. The entire thread is now pulled taut and held so by the left hand." The prevention of a duodenal fistula is a major consideration and all haste is to be avoided at this stage of the operation. No method for duodenal closure offers a more secure duodenal stump than this open method of repair.

(5) *The "Semi-Open" Technique.* Here no clamp is applied transversely to the distal duodenum, but as the bowel is severed close to the clamp to the gastric side of the line of section as in the "open" method, a series of Allis clamps catch the duodenal stump in line with its axis to give temporary compression of its coats. During the subsequent repair of the duodenal stump these clamps are removed one by one and any bleeding points ligated. This technique, like the "soft-clamp" method next delineated, is a compromise between an open and a closed treatment of the duodenum.

(6) *The Soft-Clamp Technique.* This signifies the use of a clamp with rubber-protected blades to the distal side of the line of duodenal section. Such a clamp may subsequently be left in place while a Parker-Kerr running mattress suture is applied for a first row of closure, or it may be removed at any desired time for a leisurely closure of the duodenum by the "open" method after the stomach has been dealt with, or at the time for a Billroth 1 anastomosis between the stomach and duodenal stump. The rubber-protected clamp serves, in the latter case, to give temporary hemostasis and obviates any sense of rush in the management of the duodenal stump.

(7) *The Use of the Dawson-Furniss Clamp and Pin.* This instrument and its use are shown in the self-explanatory illustration. The clamp closes the duodenal stump in the deep curvatures of the clamp blades in such a fashion that a pin may be inserted which carries a strand of suture material for the first row of closure of the open end

of the duodenum. Subsequent rows of inverting Lembert stitches and reinforcement by tabs of the omentum complete the closure.

Slight contamination of the operative field at the time of duodenal section or suture is not feared since peritonitis can seldom be attributed to this cause. Contamination with gastric contents, however, is less innocuous since the tumor-bearing segment is almost always the site of secondary infection.

Gastric resection is not safe unless at least 2 or 3 cm. of duodenal stump remain above the ampulla of Vater. If this much stump cannot be left, the thought of gastrectomy must be abandoned in favor of some form of palliative operation such as unilateral exclusion of the cancer-bearing segment.

4. *The Division of Cancerous Adhesions.* Abnormal adhesions sometimes exist between stomach and adjacent liver, omentum, or anterior abdominal wall; but for the most part these are found between stomach and transverse mesocolon, stomach and pancreas, or stomach and colon. When these are cancerous in character neoplastic extension to the neighboring structure has almost certainly occurred. Here resection will practically never prove curative and should seldom be attempted, save as a palliative procedure.

At times, however, adhesions are inflammatory in character, representing spread of infection from a deep penetrating malignant ulcer or a fungating necrotic neoplastic mass. Sometimes such inflammatory adhesions serve as an effective barrier to delay or prevent carcinomatous extension. When the surgeon feels that adhesions are only inflammatory the extirpation may be completed.

Sharp or blunt dissection is utilized, as required. Especially when dealing with the pancreas a thin layer of the adjoining structure should be pared away to be removed with the tumor-bearing segment of the stomach. An electric cautery is useful for the dissection or to control oozing surfaces which remain. The middle colic and splenic vessels are carefully guarded, but if injured must be dealt with as already described.

It should be the rule to exercise conservatism whenever stomach cancers have become attached to contiguous structures or adhesions of neoplastic nature are encountered. An exception may perhaps be warranted in case of the transverse mesocolon and transverse colon, for many definitive cures have been recorded where portions of these structures have been removed with the tumor. Whenever the structure to which the tumor was attached must be left behind, cure is exceedingly unlikely.

5. Transsection of the Gastric Walls. This stage of the operation may be concluded in a variety of ways, several of which are illustrated in Figure 5. These include: (1) the de Petz clamp technique; (2) the double clamp technique; (3) the "basting-stitch" technique; (4) the triple clamp technique; (5) the technique of "traction repair"; (6) the clamp-cut-and suture technique; (7) the "open" method; (8) tubular resection; and (9) total gastrectomy. Since each method has special points of advantage, many or all should be ready for use if needed. The value of utilizing various techniques to meet differing conditions about the pylorus and proximal duodenum becomes apparent to all who acquire flexibility in applying alternative methods. (Fig. 5.)

In severing the walls of the stomach, the rule should be to excise as much of the viscus as is consistent with safety to the patient and ease of closure, rather than to cut too close to the palpable and visible limits of the cancer. There is little basic difference between cutting across the coats of the stomach at the cardia, the body of the viscus, or the pyloric antrum. Whether to use a straight line of transsection or a curved incision, severing more of the lesser than of the greater curvature will depend upon the extent of the cancer.

Method I: The de Petz Clamp Technique. This time-saving device is placed transversely or obliquely across the curvatures of the organ and its special wheel turned to deliver the double rows of metallic clips. These remain in situ as the instrument is withdrawn and control both leakage and bleeding. With scissors, knife or cautery the stomach is now severed between the clips. Two rows of inverting stitches then invert the clips which remain on the gastric stump, securing complete closure of the organ. Non-absorbable material is employed for at least the outer row. A new gastro-enterostomy stoma must be made after such a closure; but if desired, all or a portion of the clips may later be cut away to make possible some form of end-to-side anastomosis. If the latter course is elected the first row of sutures for the gastroenterostomy is placed after properly aligning the small intestine to the gastric stump prior to reopening the viscus.

Method II: The Double Clamp Technique. This method is a close parallel to the above, save that the stomach is severed between two crushing clamps rather than between two rows of metallic clips. The gastric stump is then dealt with according to methods III, V, VI or VII, delineated below.

Method III: The "Basting-Stitch" Technique. This method requires the use of no special tools, yet provides for rapidity of execu-

tion combined with effective control of gastric leakage or bleeding. Here the tumor-bearing segment is first removed by the double clamp method; then a running, through-and-through mattress suture is applied to the stomach just proximal to the clamp which temporarily closes the cut end of the gastric stump. This suture is applied with a long straight needle which is made to pierce both surfaces of the stomach, applied alternately first from the anterior surface of the organ, then from its posterior surface. As the suturing progresses the end of the gastric stump is basted closed. The suturing begins near the greater curvature and is continued until the lesser curvature has been reached. The clamp is now removed, the gastric stump being then closed only by the basting suture. Any minute bleeding points which appear as the clamp is removed, are separately caught and tied. The same running suture is now continued as a second occluding suture, running this time from the lesser curvature until the point of origin at the greater curvature is reached; this second row is of the inverting Lambert or Cushing variety. When the point of origin is reached the suture is tied to its initial extremity, which has been left long enough for this purpose. A third row of interrupted stitches of non-absorbable material completes the closure of the gastric stump and provides additional inversion of the cut end with additional seroserous approximation. For the anastomosis to the small intestine a new gastroenterostomy is created.

Method IV: The Triple Clamp Technique. As the name implies, three clamps are here used, one toward the side of the tumor and two just to the cardiac side of this initial clamp. The two proximal clamps are applied from the two curvatures, their points meeting. The clamp toward the tumor may be of any variety, crushing or non-crushing, but at least one of the clamps toward the cardia should have rubber-protected blades, for the gastroenterostomy stoma will be made at this site as an end-to-side gastrojejunostomy, oralis partialis. The tumor-bearing segment is now removed and the portion of cut end of the stomach held within the crushing clamp is closed by the basting-stitch method, the rubber-bladed clamp near the opposite curvature being left in place until the gastrojejunal union has commenced by application of the first row of gastroenterostomy sutures. As a variant this technique may be used as follows: deal with the stomach by the two-clamp technique, then remove the tumor, leaving one clamp across the whole cut end of the gastric stump; now close the upper half of the organ by the basting-stitch, apply a rubber-bladed clamp to the remaining half along the greater curvature and remove the first clamp; conclude the operation

by making an end-to-side gastrojejunostomy, oralis partialis, at the site of the remaining rubber-protected clamp.

Method V: The Technique of "Traction Repair."   By this method the tumor-bearing segment of the stomach is used as a tractor until the gastrointestinal anastomosis has been partially completed. This technique suggested by Moynihan has special value for total or subtotal gastrectomies where suturing would otherwise be difficult on account of the high position of the abdominal esophagus or small stump of remaining stomach. The selected segment of jejunum (or mobilized duodenum) is aligned in the desired position to the posterior wall of the stomach, after turning the organ as a whole toward the left, and seromuscular interrupted mattress guy sutures are applied, followed by the first running seroserous row of gastro-enterostomy sutures.

Suitable protecting laparotomy pads are now arranged in position and the stomach and bowel are ready to be opened. Two restraining clamps are placed across the body of the stomach to prevent leakage of gastric contents; the first clamp, of any variety, is placed to the tumor-bearing side of the line of anastomosis. The second clamp must be one with rubber-protected blades or of the "soft" variety, and this is placed to the oral side of the gastroenterostomy stoma. A clamp may also be placed on the jejunum as for any union of a segment of this portion of bowel with the stomach, although this is optional. The various steps of this traction technique and all clamp arrangements are shown in Figure 5.

When the clamps are in place both the stomach and intestine are opened on a straight line 0.5 to 1 cm. from the first seromuscular suture. The second row of reparative sutures is taken as an all-layer, through-and-through running (Albert) suture. The tumor-bearing segment is now removed by severing the only remaining (anterior) wall of the stomach which is done with knife, scissors or cautery, on a line just beyond the plane of the anastomotic stoma. The anterior wall of the stomach is sutured to the remaining cut edge of the jejunum by continuing the thread of the second suture row anteriorly to constitute the third suture row in the form of an inverting Connell or Cushing suture. Clamps are now removed or loosened and if any points tend to ooze or bleed the offending vascular twigs are under-run with a suture-ligature. The anastomosis is now included by one or two rows of closely spaced stitches of non-absorbable material, making separate stitches not over ⅛ inch apart.

When a stoma of less diameter than the entire cut end of the stomach is chosen, the portion of cut edge of the stomach not needed

TRANSECTING THE STOMACH AND
REMOVING THE TUMOR—BEARING SEGMENT

DE PETZ CLAMP METHOD
AA: New gastroenterostomy stoma
if clips and cut-end are perma-
nently inverted.

DE PETZ CLAMP METHOD
SHOWING SUBSEQUENT RE-
MOVAL OF CLIPS IF EXPEDIENT

TRIPLE CLAMP METHOD

DOUBLE CLAMP METHOD

BASTING STITCH METHOD

CLAMP-CUT-SUTURE METHOD

TRACTION METHOD
FOR UNION TO JEJUNUM

TRACTION METHOD
FOR UNION TO DUODENUM

OPEN METHOD
(the cancer has already been removed)

TUBULAR RESECTION METHOD

SEMI-OPEN METHOD
Allis clamps

FIG. 5.

for the union is separately closed by one of the methods already delineated.

Method VI: The Clamp-Cut-and-Suture Technique. As the name implies this technique consists of a step-by-step or graded transsection of the stomach. The procedure is executed as follows: the tumor-bearing segment is quickly removed by the double clamp technique, leaving the gastric stump temporarily closed by a single crushing clamp. The gastric stump is now dealt with by gradually cutting away the thin segment of cut end within the grasp of this crushing clamp. For this purpose a short-nosed rubber-bladed clamp is applied parallel to the crushing clamp and a short distance therefrom; the stomach is incised between these clamps, shaving off a portion of the cut end held by the crushing clamp. This segment is repaired by the basting-stitch method, the rubber-bladed clamp is removed and the basting-stitch reinforced by a second inverting suture.

This process is repeated,—clamping, cutting and repairing succeeding portions of the cut end of the organ until the whole cut end is closed or any part not needed for a gastroenterostomy stoma has been made secure. The advantages of the method are that bleeding and gastric leakage are always under control and that no crushed margin remains on the gastric stump. The method is a cross between the open and the closed techniques for repair and aims to gain the advantages of both while falling heir to the disadvantages of neither. This clamp-cut-suture technique is also of value where the surgeon finds on surveying the cut end of the stomach, that he has cut through cancerous tissue and that an additional portion of gastric wall must be excised.

Method VII: The "Open" Method. This variant needs scant description. As the name signifies the gastric stump is dealt with by using no clamps. Multiple guy sutures, previously placed, serve to steady the remaining stomach for its subsequent repair, and temporary control of the cut end may be secured by digital compression of the gastric walls by an assistant. In a word, the method is analogous to a gastroenterostomy without clamps or to the open method for dealing with the duodenum.

Some contamination of the operative field is certain to occur during this technique which makes this procedure theoretically inferior to other methods listed; yet it is a comfort in emergencies, to know that this technique may be employed and that its routine use has been advocated by certain surgeons of wide experience.

A convenient modification of this procedure is to place a barrage of Allis clamps over the cut end of the organ, compressing anterior and posterior walls together until the cut end may be sutured; these Allis clamps are removed one by one as the suturing progresses.

Method VIII: Tubular Resection.   This is a plastic operation which serves to create a gastric stump which somewhat resembles the small intestine in diameter. The line of transection of the stomach is curvilinear. All, or almost all of the lesser curvature and its lymph node-bearing region are removed, but less of the greater curvature is sacrificed. This makes resections possible that could not be performed by severing the stomach in a straight line at right angles to the long axis of the organ. Special curved clamps have been devised for such resections. Two of these are applied and the organ is severed along the curved path between them. It is as safe and easy to sever the stomach between these clamps as between the two rows of clips applied by a de Petz clamp. The closure is completed by the basting-stitch repair.

Unfortunately, however, such special clamps are not always available or of a shape and size exactly suitable for the individual case. To obviate this difficulty, short, straight clamps may be used, aided by the clamp-cut-suture method of repair. Through-and-through cobbler's stitches of a mattress variety may also be utilized to replace the use of clamps. Here a long straight intestinal needle pierces the anterior and posterior walls of the organ, just as has been described for the basting-stitch; the direction of the needle is then reversed, creating an occluding mattress stitch as shown in the insert of Figure 5. By utilizing double rows of such occluding mattress stitches the stomach may be closed at any point or in any line, curved or straight, and the organ severed between them. After the tumor-bearing segment is severed the gastric stump is further reinforced by rows of interrupted inverting stitches.

Great flexibility of technique is soon acquired by combining any of the above listed techniques and any situation encountered may be successfully dealt with, once all of the methods have been clearly mastered.

Method IX: Total Gastrectomy.   Complete removal of the stomach has been described in previous publications. Here methods are delineated for creating a new food reservoir from anastomosed loops of intestine, for anchoring the jejunum to the diaphragm for additional security and other special expedients discussed for concluding gastrectomies of this extent. Basically, however, the removal of the entire stomach is not markedly different as to technical methods than the removal of but a portion of the organ.

Dealing with Blood Vessels within the Gastric Walls.   As the coats of the stomach are severed it is often advisable to cut first only the serous and muscular coats, leaving the mucosa temporarily intact. Here the cut muscular coats at once retract and the mucosa with its submucosal vessel promptly herniates outward in such a fashion that each vascular twig may be under-run with suture ligatures and individually ligated and severed. This prevents all bleeding.

Such a refinement of technique has distinct advantage and is a pleasure to witness, but must often be dispensed with for numerous reasons. This method for dealing with the gastric vessels is particularly applicable where the tumor is small, the stomach is easily mobilized and each step proceeds smoothly.

Resections from Left to Right (in the Cardioduodenal Direction). All of the methods for partial gastrectomy thus far reviewed have been based on severing the duodenum first and the walls of the stomach last (operating in the duodenocardial direction). In many instances, however, it proves advantageous to reverse the order and to precede the duodenal section by first severing the stomach to the cardial side of the tumor and completing the gastroenterostomy or the repair of the gastric stump before removing the cancer. This is particularly expedient when troublesome adhesions are present in the neighborhood of the head of the pancreas or when the patient represents a particularly poor operative risk.

All parts of the procedure follow essentially the same technical methods given, but the order of their execution is reversed. No rule need be laid down and the surgeon may follow his own desires and individualize his operations in choosing whether to resect from right to left or from left to right. When the gastroenterostomy is performed first, before the tumor has been completely mobilized or removed, it becomes possible to conclude the operation in its midst by excluding or segregating the tumor, leaving it in situ until a second operation (a two-stage gastrectomy).

Two-Stage Resections.   Much may be said in favor of two-stage or graded gastrectomies for selected cases. The mortalities of the two operations, however, might equal or even exceed that of the one-stage operation in any extensive series. There are certain instances when the single stage resection would certainly lead to a fatality, and it is here that the tumor must be excised in stages, if at all.

At times radiation therapy may be employed at the termination of the first operation to aid in control of the neoplasm, or even to reduce its bulk; rarely a seemingly inoperable tumor is reduced to operable proportions by this method after a period of several weeks.

Transfusions and other supportive measures are vigorously employed between stages. The chief cause for improvement proves to be an increased state of nutrition which is afforded by replacing a pyloric obstruction by an efficient gastroenterostomy.

Wedge and Sleeve Resections.   These are mentioned only to be condemned. They have little place in cancer surgery. A pedunculated polyp of doubtful malignant degeneration or an ulcer which may or may not be malignant may be successfully removed by a resection of limited extent, due to the early stage and slight spread of the neoplasm. In general, however, it is necessary and wise to remove not only the major portion of the stomach but also the associated paragastric lymph nodes and lymphatic channels.

Dissections of Unusual Extent.   Certain surgeons, notably Finsterer, have advocated removal of the entire greater omentum as part of the technique of partial gastrectomy. At times, also, more distant groups of lymph nodes may be excised; these may include the suprapancreatic nodes, and even some of the celiac or superior mesenteric nodes. Those who have followed patients subjected to resections of such unusual extent over a period of years have attested to a conviction that the increased number of long-term survivals fully justifies such extensive dissections for at least the carefully selected cases, despite a somewhat greater operative risk. In every case it is well at the conclusion of the resection to make a careful search for any enlarged lymph nodes that may have been missed; and any such should be removed.

THE RESTORATION OF GASTROINTESTINAL CONTINUITY

With the primary object of the operation completed and the cancer removed, the only remaining task is the safe reconstruction of a functionally efficient digestive tract through uniting the stump of the stomach to either the duodenum or jejunum. A veritable array of technical methods has been devised to meet this end. All of these procedures have been extensively used and their dependability tested. Thus the surgeon is free to choose at will whatever method will best meet the needs of the individual patient at the time of operation.

Although repair of the gastrointestinal tract is but the aftermath of the excision of the neoplasm a vast literature—historical, controversial, and pedantic—has arisen concerning this secondary phase of gastrectomy. During the developmental period of gastric cancer surgery this extensive literature served a vital purpose. But it has now outlived its usefulness and remains alive only to clutter and

GASTRO-DUODENAL ANASTOMOSES
(RECONSTRUCTIONS ON THE BILLROTH-I PRINCIPLE)

BILLROTH-I TECHNIC
1881
(End-to-end union)

MORISON-HORSLEY TECHNIC
1905   1926
(End-to-end with flaring duodenum)

THE HABERER-FINNEY TECHNIC
1922   1923
(Mobilizing duodenum)   (End-to-side union)

(Anterior or Posterior Side)

KOCHER TECHNIC (OBSOLETE)
1895
(Side-to-end union)

SCHOEMAKER TECHNIC
1929
(Tubular end-to-side union)

FIG. 6.

GASTRO-JEJUNAL ANASTOMOSES

Reconstructions on the Billroth-II Principle

"Choose a technic for repair only
when the cancer has been excised"

"Optional methods for reconstruction are so numerous as to ensure safe anastomosis after the performance of a gastrectomy of any extent"

Treatment of the Gastric Stump	Relation of Stoma to Colon	Name of Operation	Alignment of Jejunum	Illustration	Anatomical Description	Notes
		THE SIDE-TO-SIDE OR LATERAL ANASTOMOTIC GROUP				
A. Closed	Front	BILLROTH-II TECHNIC (1885)	ISOPERISTALTIC (Right to left)		An antecolic, side to side gastro-jejunostomy with isoperistaltic alignment of jejunum	The side to-side unions have returned to more popularity since introduction of the de Petz clamp which closes the gastric stump.
	Behind	MICKULICZ TECHNIC (1889)	ISOPERISTALTIC Right to left (originally anti-peristaltic)		A retrocolic, side-to-side gastrojejunostomy with iso-peristaltic alignment of jejunum	
		THE END-TO-SIDE OR TERMINO-LATERAL ANASTOMOTIC GROUP WITH ORALIS PARTIALIS				
B. Partially closed	Front	VON EISELSBERG TECHNIC (1886)	ISOPERISTALTIC (Lesser to greater curvature)		Antecolic, end-to-side gastro jejunostomy, oralis partialis with isoperistaltic jejunum.	The oralis partialis principle allows more of lesser curvature than of greater to be excised
	Behind	HOFFMEISTER-FINSTERER TECHNIC (1905) (1913)	ISOPERISTALTIC (Lesser to greater curvature)		Retrocolic, end-to-side gastro jejunostomy oralis partialis with isoperistaltic jejunum	
		THE END-TO-SIDE OR TERMINO-LATERAL ANASTOMOTIC GROUP WITH ORALIS TOTALIS				
C. Open (used for anastomosis)	Front	KRÖNLEIN-BALFOUR TECHNIC (1887) (1916)	ISOPERISTALTIC (Lesser to greater curvature)		Antecolic, end-to-side gastro jejunostomy, oralis totalis with isoperistaltic jejunum	The original oralis totalis procedure.
		MOYNIHAN-II TECHNIC (1922)	ANTIPERISTALTIC (Greater to lesser curvature)		Antecolic, end-to-side gastro jejunostomy oralis totalis with antiperistaltic jejunal alignment.	Requires shorter jejunal loop. A popular method
	Behind	REICHEL-POLYA TECHNIC (1911) (1907)	ISOPERISTALTIC (Lesser to greater curvature)		Retrocolic, end-to-side gastro jejunostomy oralis totalis with isoperistaltic jejunum	Has been more used than any other gastro jejunal union
Special Technics	All are relatively minor variants of above basic operations	ROUX TECHNIC (1893)	An "en-y" anastomoses		Jejunum severed. Distal limb sutured to cut end of stoma Proximal limb sutured to side of distal jejunum.	Complicated suturing seldom used but valuable for rare cases with adhesions.
		LAHEY TECHNIC (1932)	Mobilization		of terminal duodenum added to a Reichel-Polya operation	Reputed to lessen complications at jejunal angle
		MOYNIHAN-I TECHNIC	Another "en-y"		variant. End-to-side.	Same usefulness and disadvantages as Roux operation
		FINSTERER TECHNIC	Often adds		removal of whole great omentum to operation	More cures claimed for the wider resection (HOFFMEISTER-FINSTERER CLOSURE)
		BRAUN ANASTOMOSIS	A supplementary		Jejuno – Jejunostomy may be added to any technic	Avoids the "viscious cycle"
		DUBOURG TECHNIC (1898)	An antecolic		side to side gastro-jejunostomy with new stoma made on the POSTERIOR wall of the stomach	

FIG. 7.

obstruct the subject, needlessly burdening the mind of student and inexperienced operator.

The accompanying charts (Figs. 6 and 7) on gastrectomy summarize this subject. Here the many available operations are listed by their eponymic names, basically classified, and dates recorded when first proposed or applied. It proves difficult, however, for the surgeon even to memorize all of these methods or to retain in mind their historical interrelationships upon which the terminology is based.

Yet the operator should be consoled rather than disturbed by this multiplicity of methods. For he is thereby furnished with a wide range of optional techniques from which to draw for any specific need. Since each method in itself is essentially easy to execute, the complexity is found to reside chiefly in an intellectual mastery of so large a list of separate measures. This difficulty is obviated by focusing attention on the few fundamental principles which underlie all points of differentiation. Time spent in acquiring a thorough working knowledge of at least these fundamental principles of repair will be amply repaid in practical work. The more complete the intellectual mastery of this assortment of technical variables, the greater the confidence and ease in meeting unexpected complications and unusual problems while operating. How could any specific technique be successfully applied on the human which could not be accurately described or sketched from memory? It is the object of the ensuing portion of this article to present all of the useful techniques with as much simplicity, practicability, and ease of memorization as can be accorded this topic without sacrifice of accuracy of detail.

1. GASTRODUODENAL RECONSTRUCTION (THE BILLROTH I PRINCIPLE)

The earliest gastrectomies were terminated by suturing the open end of the gastric stump to the open end of the duodenum (terminal or end-to-end anastomosis). Every repair which makes use of the duodenum for the anastomosis is now referred to as a reconstruction on the Billroth I principle. Anastomoses which utilize the jejunum are collectively termed repairs on the Billroth II principle.

Five types of gastroduodenal repairs are the following:

1. End-to-end gastroduodenostomy (original Billroth I technique).

2. End-to-end gastroduodenostomy with flare of the duodenum (the Morison-Horsley technique).

3. End-to-end gastroduodenostomy with tubular reconstruction of the stomach (the Schoemaker technique).

4. End-to-side gastroduodenostomy (the Haberer-Finney technique).

5. Side-to-end gastroduodenostomy (the Kocher technique).

It should be noted that in these self-definitive names the initial word in the compound designations always refers to the stomach and the last word, to the duodenum. Figure 6 illustrates all of these variants of the Billroth I principle.

1. *End-to-end or Termino-Terminal Gastroduodenostomy* (*Billroth I Operation*). In this technique the open end of the duodenum may be sutured to the region of the lesser curvature (oralis superioris), to the region of the greater curvature (oralis inferioris), or to the mid-portion of the cut end of the stomach (oralis intermedius). The so-called "fatal angle," which is a point where two suture lines meet (Figure 6), has proved a deterrent to the use of this method for repair. Postoperative leakage at this point occurred with discouraging frequency in the early days of gastric surgery, leading to fistula formation, peritonitis, and often to death of the patient. Recent improvements in details of gastrointestinal suturing have gone far toward removing this danger. A special mattress stitch for use at this angle to ensure broad peritoneal approximation and added security is illustrated in Figure 6. Interrupted stitches of non-absorbable materials also add to safety of the repair and are essential in dealing with cancers. The portion of the stomach not used for the anastomosis, also may be covered by suturing the lateral wall of the duodenum over this part of the gastric stump. Yet it was largely the insecurity of this deadly angle that encouraged development of the remaining forms of gastroduodenostomy.

2. *End-to-End Gastroduodenostomy with Flare of the Duodenum* (*the Morison-Horsley Technique*). Rutherford Morison was the first to suggest splitting the duodenal stump in its long axis for a short distance (2 to 5 cm.) to widen or flare the opening of its cut end and bring the diameter of the duodenal stump into closer approximation to the diameter of the cut end of the stomach. Horsley later popularized the method.

3. *End-to-End Gastroduodenostomy with Tubular Reconstruction of the Gastric Stump (the Schoemaker Technique)*. This variant chiefly concerns the line of resection of the cancer. In place of a straight transverse line of section of the stomach a curved line is employed which removes considerably more of the lesser than of the

greater curvature. Special curved clamps may be used to accomplish this result, or sutures of special type may be employed.

Advocates of this method point out that cancer spreads with greatest frequency along the nodes of the lesser curvature, making this region a marked danger point with regard to recurrence or incompleteness of removal. When the stomach is reconstructed as a long tube from the greater curvature this may be more easily united to the duodenum without tension and the two viscera fit one another more perfectly.

The method is an adaptation to gastroduodenal repair of the principle of oralis partialis reconstruction which had earlier been advocated by Von Eiselsberg for gastrojejunal anastomosis. Technical difficulties in sectioning the stomach and in subsequent suturing make this method applicable chiefly to selected cases with subtotal implication of the stomach by the carcinoma. It is possible by this method to close the end of the gastric tube, making a side-to-end repair, or to leave it open for an end-to-side gastroduodenostomy, as well as to unite the two organs in the end-to-end or terminal fashion.

4. *End-to-side or Terminolateral Gastroduodenostomy (the Haberer-Finney Technique).*   Here the cut end of the stomach is united to the side of the duodenum. To effect this union the duodenum is extensively mobilized before the cancer is removed. A bloodless fold of peritoneum, passing from the second portion of the duodenum to the posterior parietal wall of the abdomen, is cut. This incision is made parallel to and just beyond the convex curvature of the duodenum. The duodenum is then freed by blunt dissection from its posterior attachments, which restores its embryologic position as a midline organ with a posterior mesentery (Kocher mobilization of the duodenum). This resulting wide mobility of the bowel sometimes makes possible its anastomosis even to the abdominal esophagus.

After removal of the gastric cancer the cut end of the duodenum is closed as previously described and the mobilized viscus united to the cut end of the stomach by an end-to-side union. The usual four-layered type of gastrointestinal suturing is employed, with interrupted stitches for at least the outer rows. After repair the ampulla of Vater lies opposite the anastomotic stoma, but this theoretical objection seems to cause no difficulty in actual use of the method which has proved satisfactory for selected cases.

5. *Side-to-End of Lateroterminal Gastroduodenostomy (Kocher Technique).*   This modification of the Billroth 1 principle is now chiefly of historical significance and but seldom applied. Here the cut end of the stomach is entirely closed and the open end of the

duodenum sutured to the side of the organ in a new anastomotic opening (a side-to-end repair). The resulting stoma is small and the suturing more difficult than in the previously described and more popular methods.

II. GASTROJEJUNAL RECONSTRUCTION (THE BILLROTH II PRINCIPLE)

The appended comprehensive table summarizes for reference and study all techniques for restoring the continuity of the gastro-intestinal tract which utilize the jejunum for the anastomosis. (Fig. 7.) These many methods are termed, respectively, the Reichel-Pólya operation, the Krönlein-Balfour operation, the Moynihan operation, the Mikulicz operation, etc. Yet they should not be viewed as actually constituting separate operations, since in reality they are but minor variants of the single surgical procedure, gastric resection.

The psychological attitude of the surgeon on this point has significance, for it is requisite in the midst of apparent complexity to grasp the essential simplicity of the subject. This simplicity resides in the fact that as previously emphasized, only one method is applied to any individual patient and each proves relatively easy to perform. The availability of so wide a range of variants possesses great practical value. It is unwise for the surgeon to depend implicitly upon but a single procedure, as so often is done by the novice, for this leaves the operator totally unprepared to deal with the protean manifestations of gastric cancers. Increasing experience discloses that several techniques must be used as circumstances indicate; and the veteran surgeon in this field will make use of all of the most important and basic procedures so laboriously developed by his surgical forebears. The situation is one in which all confusion must be eliminated before the peritoneal cavity is opened, for confusion in the *preoperative* understanding of these optional methods, if unconquered, is certain to result in confusion *in the midst of the operative work*, at the expense of the patient's interests, perhaps of his life. Unless the surgeon is fully prepared to deal with any type of tumor encountered he will find that the degree of preparation adequate for managing a small prepyloric cancer often will not suffice for the type of cancer actually present. The only dependable preparation for gastric cancer surgery is that attained by penetrating all superficial intricacies of the subject and gained by acquiring ability to employ at will any technical method most advantageous to follow in the specific case at hand.

The many variants of the Billroth II principle are all based on six fundamental points, three of which are of major importance and three of lesser significance. The following are the major points: (1) Whether the anastomosis is made in front of or behind the transverse colon (anterior vs. posterior gastrojejunostomy). (2) Whether the open end of the stomach is used for the repair or this is closed and a new gastroenterostomy stoma is created (end-to-side vs. side-to-side union). (3) What direction is given to the jejunal loop in its alignment with the stomach (isoperistaltic vs. antiperistaltic anastomosis). The secondary and remaining considerations are as follows: (4) Whether the entire cut end of the stomach or but a portion is utilized, in the end-to-side repairs (oralis totalis vs. oralis partialis). (5) Whether or not a supplementary jejunojejunostomy is added to the gastrojejunostomy. And (6) Whether the jejunum is used in the form of a loop or is severed and the anastomosis made of the "en-Y" type.

The appended systematizing summary table of all the specific methods not only gives details as to each method but also allows them to be studied in groups according to points of similarity and difference based on these six fundamentals. There is good reason why the surgeon should fully investigate these interrelationships of the optional methods. For, during any gastric resection a series of important decisions must be reached before the operation can progress; for example, to make the anastomosis to the jejunum; to create a retrocolic stoma; to utilize the whole cut end of the stomach for the repair, etc. As these decisions are made, this automatically reduces the number of possible techniques from the original score or more to an ever decreasing field of possibilities. Under any given set of circumstances based on the six fundamental points (and these necessary decisions) only two, three, or four optional procedures remain for concluding the gastrectomy. This makes clear why the interrelationships and groupings of these techniques are almost as important as is knowledge of the separate methods themselves. This significant consideration is readily comprehended as the chart is perused and ensuing paragraphs are studied.

SIDE-TO-SIDE OR LATERAL GASTROJEJUNAL REPAIR

When the cut end of the stomach is entirely closed a new gastroenterostomy stoma must be created. Two important techniques are then available for completing the operation: namely, (1) the original Billroth II technique and (2) the Mikulicz technique. These, with the exception of two or three very minor variants thereof and the

seldom used "en-Y" type of anastomosis, exhaust the possibilities for reconstruction when the open end of the gastric stump is not utilized. This greatly simplifies problems of memorization of methods and of actual reconstruction and means that when a de Petz clamp is used or the stomach stump is otherwise closed, the surgeon need not consider the total of twenty or thirty possible methods for closure, but may then confine his attention to the two techniques named above and their minor variants.

1. *The Original Billroth II Technique.*   The first operation using jejunum for the gastroenterostomy after resection of a cancer was performed in 1885 by Billroth, four years after this pioneer in gastric surgery executed the first successful gastrectomy (1881). The gastrojejunal union was made in front of the colon, an antecolic repair. The gastroenterostomy was made several days prior to the extirpation of the cancer because the patient was too ill at the first operation to withstand resection of the carcinoma. Postoperative conditions were so favorable that a second operation was decided upon and the tumor successfully excised. Since a functioning gastroenterostomy was already present, the cut ends of both duodenum and stomach were simply closed by inversion. These facts make clear that the original Billroth II technique was an antecolic, side-to-side, or lateral gastrojejunostomy, the direction of the jejunal loop being that shown in the illustration (Fig. 7), a so-called "isoperistaltic" alignment. While these are the details of the specific Billroth II *technique*, the entire principle of utilizing the jejunum for the repair is now referred to as the Billroth II *principle* of closure, and of this principle there are many separate specific techniques which have been applied as modifications by numerous surgeons.

2. *The Mikulicz Technique.*   Mikulicz soon performed behind the transverse colon exactly what Billroth had done in front of the colon. Thus this operation (1889) was a *retrocolic,* side-to-side, or lateral gastrojejunostomy, with isoperistaltic alignment of the jejunal loop. An historical footnote on the development of gastroenterostomies reveals why Billroth went in front of the colon for his original operation and Mikulicz used the posterior route, for posterior gastrojejunostomy for benign lesions was developed in the interval between these two operations for cancer.[1] A supposed advantage of

[1] The initial gastrojejunostomy was performed for a benign lesion of the stomach and was devised by Anton Wölfler, an assistant of Billroth in his Vienna clinic. The year was that of the first resection, 1881; but gastrojejunostomy was never used for cancer until performed by Billroth himself in 1885, as above related.

The first *posterior* gastrojejunostomy is commonly credited to von Hacker who performed the operation late in 1885. It is historically true that Courvoisier had made

the posterior route is that less distance is required between the duodenojejunal angle and the gastrojejunostomy stoma, that is, the jejunal loop is shorter, which is reputed to lessen postoperative complications. A disadvantage is the obvious necessity of going through the transverse mesocolon to effect the union.

It is of more than passing interest to note that while the above two techniques were among the earliest methods for repair after the resection of cancers of the stomach and although they were soon superceded for the most part by other techniques which utilized the open end of the gastric stump for the anastomosis, there has been a decided tendency in recent years to revert to these original procedures. This is accounted for by the development of such special suturing instruments as the de Petz clamp which automatically and quickly closes the entire end of the gastric stump. The so-called "basting-stitch" method for dealing with the stomach also closes the gastric stump and lends itself to a subsequent lateral or side-to-side repair. Unless such a side-to-side union is made, once the gastric stump has been completely closed, it becomes necessary to cut away a portion of the sutures or metallic clips after they have been put in place, which consumes time and represents a reduplication of effort. For this reason it is usually easier to conclude the operation by use of either the original Billroth II technique or the Mikulicz technique. The optional little-used and minor variants are shown in Figure 7.

END-TO-SIDE OR TERMINOLATERAL GASTROJEJUNAL REPAIR

The first of all the modifications of the original Billroth II technique was that of Krönlein who advocated that the whole cut end of the gastric stump should be made a part of the gastrojejunostomy stoma (1887). Such terminolateral or end-to-side unions, which use the whole cut end of the stomach have since become known as unions on the *basic plan of Krönlein*. The actual *technique* of Krönlein was an antecolic end-to-side gastrojejunostomy, oralis totalis (using the whole end of the stomach), with isoperistaltic jejunal alignment. The many additional techniques which use the whole cut end of the stomach may be seen at once by glancing at Figure 7 where all methods are grouped to bring out similarities and points of distinction. The practical point is that the present-day operator is free

---

an anastomosis behind the colon over three years earlier, but this had attracted no attention and was performed by an intricate method which never gained popularity and is now obsolete (going first through transverse mesocolon, then through gastrocolic ligament for a union to the front of the stomach).

to elect at will whether he will perform a lateral union (side-to-side) or choose a terminolateral union (end-to-side); that is, he may close the gastric stump and make a new anastomosis, or use the open end of the stomach as part of the stoma, as circumstances of any specific case indicate.

It was at once felt that the resultant stoma in an end-to-side anastomosis might prove too large, since it so far exceeded the diameter of the normal pylorus; and it was proposed that a portion of the cut end of the stomach be closed and only the remainder, near the greater curvature, be employed for anastomosis to the jejunum. The first to execute this modification was von Eiselsberg (1888) and this oralis partialis principle for gastrojejunostomies became known for all techniques which made use of it as operations on the *basic plan of von Eiselsberg*. Just as the modern surgeon is free, then, to unite the stomach to either the duodenum (Billroth I principle) or to the jejunum (Billroth II principle); and just as he is free to close the gastric stump and make a side-to-side union or to leave this open and make an end-to-side union; so is he free to use the whole end of the organ (operating on the plan of Krönlein, oralis totalis), or to utilize only a part of the cut end of the organ after closing the region of the lesser curvature (operating on the plan of von Eiselsberg, or making an oralis partialis).

The early sequence of events, which is now of importance only as a means for clearly understanding basic points of anatomic variation between optional methods for repair and of memorizing accurately the eponymic terminology which tends to be retained, is seen to have been as follows: In making gastrojejunal repairs Billroth (1885), Krönlein (1887) and von Eiselsberg (1888) all worked in front of the colon—the first doing a side-to-side anastomosis, the second doing an end-to-side union with oralis totalis, and the third doing an end-to-side union with oralis partialis. These early workers were followed by Mikulicz (1889) who went behind the colon to do the initial retrocolic operation for gastric cancer. The surgeon may now follow any or all of these procedures in his own cases, remembering the names of the originators of the techniques if he so desires, or brushing these aside to describe his operation on a purely anatomic basis.

A. *Antecolic End-to-Side Repairs.* The important eponymic techniques for gastrojejunostomies using the open end of the stomach in front of the transverse colon are but four in number. The first two of these are identical and all but one have already been described in principle.

1. *The Krönlein Technique.* This is an antecolic, end-to-side gastrojejunostomy, oralis totalis, with isoperistaltic jejunal alignment, as amply discussed. It is the first operation to use the whole cut end of the gastric stump for the union to the jejunum, and was the first modification to be made of the Billroth II principle.

2. *The Balfour Technique.* This is identical with the above technique and represents a late popularization of the Krönlein method. The long period (over thirty years) after the work of Krönlein in which this technique was almost entirely dropped in favor of various retrocolic repairs justified its revival as almost a new technique (an antecolic end-to-side gastrojejunostomy, oralis totalis, with isoperistaltic alignment of the jejunum). Confusion is avoided by giving this procedure its proper name as a compound designation, *the Krönlein-Balfour operation,* honoring both originator and popularizer of this most useful method. Balfour also advocated a supplementary lateral anastomosis of the ascending and descending limbs of the jejunal loop for selected cases.

3. *The Moynihan Technique.* This differs from the above two procedures only with regard to the direction of the alignment of the jejunum which in this case is antiperistaltic. This means that the distal or efferent limb of the jejunal loop leaves the *lesser* curvature of the stomach and passes toward the *right* rather than leaving the greater curvature and passing toward the left as is the case with the isoperistaltic unions. This topic which has always proved puzzling to the student is discussed under a separate heading where the distances from duodenojejunal angle to anastomotic stoma for the various techniques are also discussed. The surgeon should grasp the principle involved rather than arbitrarily remember a set distance for each specific operation. For the Moynihan method the distance is approximately 4 to 6 inches (10 to 15 cm.). The Moynihan method, then, is an antecolic, end-to-side gastrojejunostomy, oralis totalis, with *antiperistaltic* jejunal alignment. Since Moynihan also described an "en-y" type of anastomosis the procedure has at times been called a Moynihan II method, but this refinement is needless since all gastroenterostomies "en-y" are so seldom performed (see subsequent heading on "en-y" unions).

4. *The von Eiselsberg Technique.* This has already been defined and is an antecolic, end-to-side gastrojejunostomy, oralis partialis, with isoperistaltic alignment of the jejunum. It represents the first application of the oralis partialis plan of anastomosis to gastrojejunal repairs.

Whenever the surgeon plans to unite a loop of the jejunum to the open end of the stomach in front of the colon, then, the above three methods are his only options. This knowledge, combined with that of "en-y" operations, later discussed, and of the Billroth II technique for an antecolic union with the closed end of the stomach exhaust the whole subject of antecolic unions and reveal the essential simplicity of this topic of optional methods.

B. Retrocolic End-to-Side Repairs. Only two ways are available for uniting the side of a jejunal loop to the end of the stomach retrocolically. One of these is to use the whole cut end of the stomach, which has had two prominent advocates; the other is to use but a portion of the cut end (posterior application of the partialis stoma plan), which also has had two prominent advocates.

1. *The Reichel Technique.* Reichel performed behind the colon what Krönlein had done in front of the colon, namely, used the whole cut end of the gastric stump for the repair. The operation is, therefore, a retrocolic, end-to-side gastrojejunostomy, oralis totalis, with isoperistaltic alignment of the jejunal loop. The procedure was described in 1907 (Reichel) and later popularized by Pólya (1911).

2. *The Pólya Technique.* This is identical with the above method, hence its proper designation should be *the Reichel-Pólya operation.* No method of reconstructing the gastrointestinal tract after resections for cancer has been so widely employed.

3. *The Hoffmeister Technique.* This is a retrocolic utilization of a part of the cut end of the stomach for the reconstruction. It represents an operation behind the colon corresponding to that of von Eiselsberg's operation in front of the colon. More of the lesser than of the greater curvature is resected, the region of the lesser curvature is then repaired, and the anastomosis to the jejunum is made with the remainder of the cut end of the stomach nearer the greater curvature. The full description of the operation is: a retrocolic, end-to-side gastrojejunostomy, oralis partialis, with iso-peristaltic jejunal alignment.

4. *The Finsterer Technique.* This represents chiefly a popularization of the Hoffmeister technique just described. Hoffmeister published his operation in 1905 while Finsterer described his technique in 1911. Both are retrocolic applications of the oralis partialis principle of repair after extensive removal of the lesser curvature. Finsterer adds removal of the entire greater omentum during the stage of excision of the tumor to many of his operations. The proper designation for this retrocolic method of repair should be by the compound name, *the Hoffmeister-Finsterer operation.*

The Alignment of the Jejunal Loop.   The loop of jejunum may be attached in line with the long axis of the stomach, transverse to this long axis which means from curvature to curvature, or obliquely. When the alignment follows the long axis of the stomach one limb of the loop runs toward the patient's left and the other toward his right. Since the first portion of the jejunum normally travels downward and *toward the left* within the peritoneal cavity, it is said that the jejunal loop has been attached in a "normal" or "isoperistaltic" direction when the distal limb takes this same direction, namely, downward and toward the left. The term has become fixed in surgical literature but has always excited complaint. If the prefix "iso" referred to the jejunum only and meant "in the same direction as usual" the matter would present little difficulty; but *isoperistaltic* means, literally, with peristaltic wave traveling in the same direction in the stomach as in the jejunum after the alignment. It is not easy to show that this is ever the case, no matter how the alignment is made. Yet since no better term has ever been proposed this terminology continues to be employed but continues to prove confusing.

The best practical solution is to accept arbitrarily as a definition of an isoperistaltic alignment, a situation where the distal or efferent limb of the jejunum leaves the greater gastric curvature traveling downward and toward the patient's left, in approximately its normal course. An antiperistaltic union, per contra, means one in which the distal limb of the jejunum either passes toward the *right* or leaves the *lesser curvature* of the stomach and passes toward the right, which distorts it from the normal course. There is no trouble in memorizing these alignments when it is kept in mind that the single method of gastrojejunostomy now in use which calls for an antiperistaltic alignment is the Moynihan technique (sometimes called the Moynihan II method). While the original gastrojejunostomy which Wölffler performed for benign lesions of the stomach was also an antiperistaltic alignment, this author changed soon afterward to the isoperistaltic alignment.

The major point in attaching the small bowel to the intestine is to ensure that the gastric contents enter the distal or efferent limb after the reconstruction rather than to be forced backward toward the duodenum, producing the dreaded vicious circle. The distance of anastomosis from the duodenojejunal angle has a bearing on this point. For isoperistaltic end-to-side repairs in front of the colon (Krönlein-Balfour; von Eiselsberg) the proximal limb of the jejunal loop must be long, since it travels from the duodenojejunal angle over the colon and the greater omentum to the lesser gastric curva-

ture before the anastomosis begins; here at least 10 to 15 inches (25 to 38 cm.) of proximal jejunum are needed between the lesser curvature and the end of the duodenum (the angle). The exact distance depends somewhat on the amount of stomach removed, the mobility of the jejunum and the amount of fat in the transverse mesocolon and omentum. With such long limbs to the loop a supplementary jejunojejunostomy (Braun anastomosis) is often expedient, made just below the plane of the transverse colon.

For the antecolic repair of Moynihan, (which is antiperistaltic in alignment), the proximal jejunal limb need only be 4 to 6 inches (10 to 15 cm.), since the duodenojejunal angle and the greater gastric curvature lie at a relatively close distance from one another.

For such retrocolic anastomoses as the Reichel-Pólya or Hoffmeister-Finsterer techniques the distance from the duodenojejunal angle to the lesser curvature requires a jejunal length of about 4 to 5 inches (10 to 13 cm.), although this may be some what increased if desired.

Regardless of the exact distances advised as proper, the surgeon should be on the alert at this stage of the operation to see for himself that the length of the proximal limb of the jejunum is neither too long nor too short. At the termination of the gastrointestinal suturing he must also decide with each individual case whether or not a supplementary jejunojejunostomy should be added to the gastrojejunostomy.

A technical variant known as *the Lahey technique* may appropriately be described here. This consists of mobilizing the duodenojejunal angle before an anastomosis is made, freeing the terminal duodenum. This simplifies the matter of distances, for by this mobilization the terminal duodenum and first part of the jejunum are made to lie entirely above the attachment of the transverse mesocolon to the posterior abdominal wall. This is reported by the originator to reduce postoperative complications, since after a posterior gastrojejunostomy by this method only the distal or efferent limb of the jejunal loop will traverse the hole made in the transverse mesocolon. The Lahey operation is terminated as a retrocolic, end-to-side union, oralis totalis, with isoperistaltic alignment of the jejunal loop. In a word, it is a Reichel-Pólya operation supplemented by preliminary mobilization of the duodenal angle. Details of the technique include: (a) making the hole in the transverse mesocolon just to the left of the root of the ligament of Treitz, guarding carefully against injury to the middle colic artery; (b) bringing an ample loop of jejunum through this rent to allow for a

Reichel-Pólya anastomosis, with no tension on the proximal jejunal limb after the repair is complete and the small gastric stump has retracted high into the left hypochondrium; (c) dividing the ligaments of Treitz, from its lowest insertion into the jejunum, up to its origin in the mesenteric root. "This permits the mobilization of the uppermost part of the jejunum, so that the proximal loop of jejunum now anastomosed to the stomach can be passed up through the slit made in the transverse mesocolon, and in this way the entire proximal loop of jejunum is brought above the mesocolon and is excluded from the greater general peritoneal cavity." At the conclusion of the operation the slit in the transverse mesocolon is snugly sutured about the single limb of bowel (distal limb of jejunum) which traverses this structure.

ANASTOMOSES "EN-Y"

The only remaining group of gastroenterostomies to be described is that in which the jejunum is severed before being aligned to the stomach rather than being aligned as a loop. The best known "en-y" method is *the Roux technique* (1893) in which the distal segment of the jejunum, after the bowel has been severed, is united to the open end of the gastric stump as an end-to-side union retrocolically; a supplementary anastomosis is made between the end of the proximal segment of the jejunum and the side of the distal segment, at a convenient point (end-to-side jejunojejunostomy), which unites these segments of bowel in the form of a "y." (Fig. 7.) It is obvious that the cut end of the bowel can be anastomosed to the stomach not only retrocolically, but also in front of the colon; the union may be end-to-end, end-to-side, side-to-end, or side-to-side, which produces a wide variety of optional closures, the names of which need not be retained. "En-y" anastomoses are seldom employed save where extensive carcinomatous adhesions are encountered which include the small intestine and make it difficult to deliver a jejunal loop to the region of the stomach. Suturing details are more complicated and, in general, the method is one which is held in reserve but proves invaluable in rare instances.

GASTROINTESTINAL SUTURING

The four-layer gastroenterostomy suture methods well known to every surgeon are employed in this second phase of repair after gastric resection; but at least the outer two rows should be of a non-absorbable material, and interrupted stitches are decidedly preferable for these outer rows and for all reinforcement sutures.

The omenta should be freely drawn upon to cover suture lines for final support. Stitches should be especially close ($\frac{1}{8}$ inch) in suturing the walls of the cancerous stomach because of delayed healing. These are snugly drawn but not made tight enough to cause necrosis. Gastroenterostomy stomas after resection for gastric cancer should be of wide diameter to allow for contracture or for invasion in cases of recurrence ($2\frac{1}{2}$ to 3 inch stoma). The longitudinal incision in the jejunum is always made a little shorter than that in the stomach, since jejunal walls are thinner and more distensible. The rent in the transverse mesocolon after retrocolic unions is stitched to the gastric side of the anastomosis if this will cause no distortion; otherwise it is lightly caught about the two limbs of the jejunum traversing this structure.

Gloves are changed and clean drapes applied at the end of the gastroenterostomy repair and before the abdominal wall is repaired. "Rough handling, rough wiping, in fact roughness of any kind is to be scrupulously avoided; the light hand, here as always within the abdomen, is essential."

Drains are seldom required, but a gutta percha strip or split tube may be inserted if gross contamination has occurred or if postoperative insecurity is feared. Postoperative fistula formation, particularly at the duodenal stump, is a serious complication and must be rigorously guarded against by meticulous care in placing sutures. Fear of an operative death because of time necessity for scrupulous accuracy in the placement of stitches should never be commensurate with the fear of any postoperative insecurity. Slight contamination of the peritoneal cavity and intraperitoneal pads at the time of operation, likewise, has none of the danger that is attached to the slightest degree of postoperative leakage.

CHOICE OF METHODS

There are few absolute indications for the selection of any particular technique. The choice of methods is largely a matter of training, preference, and individualization. When a suturing instrument is employed a side-to-side union is usually the simplest method for closure; but there should be no hesitation in cutting away a portion of the temporary hemostatic clips in order to select some form of end-to-side procedure. If the whole viscus is involved a total gastrectomy is the only possible curative operation; when there is subtotal implication of the stomach it is often possible to cope with the situation by a curved line of section of the stomach, which

necessitates closure by the von Eiselsberg, the Hoffmeister, the Finsterer, or the Schoemaker technique.

The Billroth II principle of repair is to be preferred to the Billroth I plan of anastomosis except for small tumors where the duodenum can be attached to the gastric stump without undue tension. To employ the Billroth I principle often tempts the surgeon to try to conserve more stomach than is consistent with safety. It should be a rule to remove as much of the stomach as can be accomplished without making subsequent gastrojejunostomy too difficult.

"En-Y" anastomoses are used only under the demand for such an unusual procedure as dictated by the presence of complicating adhesions. The question of supplementary jejunojejunal anastomosis must be decided in each individual case at the conclusion of the gastroenterostomy, but is added when it appears that there may be either anatomic or physiologic obstruction to the free passage of biliary and duodenal fluids into the small intestine beyond the gastroenterostomy stoma. The most popular type of reconstruction after resections of gastric cancers has been the Reichel-Pólya technique, or its antecolic counterpart, the Krönlein-Balfour operation.

SUMMARY

The purpose of gastrectomy for cancer is a wide removal of all carcinomatous tissue within the stomach and juxtagastric lymphatics. The two divisions of the operation (A) extirpation of the cancer and (B) restoration of gastrointestinal continuity should be sharply differentiated. Selection of the technique for anastomosis should as a rule be delayed until the stage of excision has been concluded. No single method for repair proves suitable for all cases. Flexibility of technique is a prime requisite in gastric cancer surgery. The score of optional methods available provide ample means for safe reconstruction of the digestive tract no matter how extensive the carcinomatous invasion.

Alignment of the parts without tension, meticulous care in suturing to ensure broad peritoneal approximation, the use of nonabsorbable suture materials for the outer rows of sutures and free use of interrupted stitches, are essentials that cannot be disregarded with impunity.

The entire subject of gastrointestinal repair after resections for carcinoma has long been considered complex and difficult; but much of the apparent complexity is pedantic. Relative simplicity is

obtained by focusing attention on the six fundamental anatomic points which underlie similarities and differences between all methods. The operations should be anatomically described for postoperative records, whether or not eponymic names are added.

Palliative operations for stomach cancer offer little prolongation of life. In a disease so rapidly fatal without excisional surgery a considerable operative mortality may be faced with equanimity. An attempt should be made to remove the cancer in one or two stages wherever there appears to be reasonable hope of success. In competent hands partial gastrectomy for cancer has proved to be "an operation without undue risk but an operation of generous promise."

REFERENCES

1. BALFOUR, D. C. Restoration of gastro-intestinal continuity by means of gastro-jejunostomy following partial gastrectomy for cancer of the pyloric end of the stomach. *Surg., Gynec. & Obst.*, 25: 473–477, 1917.
2. BALFOUR, D. C. A method of carrying out two-stage operations for carcinoma of the stomach. *J. A. M. A.*, 90: 1936–1938, 1928.
3. BICKHAM, W. S. Operative Surgery, Vol. 4. Philadelphia, 1924. W. B. Saunders Co.
4. BINNIE, J. F. Operative Surgery, 8th ed. Philadelphia, 1921. Blakiston's Sons & Co.
5. BILLROTH, T. Offenes Schreiben an Herrn Dr. L. Wittelshoefer. *Wien med. Wchnschr.*, 31: 161–165, 1881.
6. BRAUN, H. Zur Technik der Naht bei vershiedenen Operationen am Magen und Darm. *Deutsche med. Wchnschr.*, 17: 3–4, 1891.
7. BRAUN, H. Ueber die Gastroenterostomie und gleichzcitige Enteroanastomose. *Zentralbl. f. Chir.*, 19: 102, 1892.
8. CLAIRMONT, P. Bericht ueber 258 von Prof. v. Eiselsberg ausgefuehrten Magen-operationen. *Arch. f. klin. Chir.*, 76: 180–322, 1905.
9. CREITE, O. (Braun) Beitraege zur Chirurgie des Magencarcinoms. *Deutsche Ztschr. f. Chir.*, 87: 275–309, 1907.
10. CZERNY. Therapie der krebsigen Stricturen des Oesophagus, des Pylorus und des Rectum. *Berlin. klin. Wchnschr.*, 34: 762, 779, 1897.
11. DOUGLAS, J. Operations upon the Stomach. New York, D. Appleton & Co.
12. DOYEN, E. Traitement des affections de l'estomac et du duodénum. Paris, 1895. Rueff et Cie.
13. EISELSBERG, A. VON. Ueber die Magenresectionen und Gastroenterostomieen in Prof. Billroth's Klinik March, 1885 to October, 1889. *Arch. f. klin. Chir.*, 39: 785–844, 1889.
14. EISELSBERG, A. VON. Unsere Erfahrungen mit der Behandlung des Magen-und-duodenal Ulcus. *Wien. klin. Wchnschr.*, 39: 709–713, 1926.
15. FINNEY, J. M. T. A new method of gastroduodenostomy. *Tr. South. Surg. A.*, 36: 576–578, 1923.
16. FINNEY, J. M. T., and HANRAHAN, E. M., JR. Gastric ulcer. In Lewis' Principles of Surgery, vol. 6, chapter 6, Hagerstown, Md., 1929. W. F. Prior Co.
17. FINNEY, J. M. T., and RIENHOFF, W. F. Gastrectomy. *Arch. Surg.*, 18: 140–162, 1929.
18. HABERER, H. VON. Terminolaterale Gastroduodenostomies bei der Resektions. Methode nach Billroth 1. *Zentralbl. f. Chir.*, 49: 1321–1326, 1922.
19. HABERER, H. VON. Meine Technik der Magenresektion. *München. med. Wchnschr.*, 80: 915–921, 1933.

20. HACKER, VON. Zur Casuistik und Statistik der Magen-Resektionen und Gastro-enterostomieen. *Arch. f. klin. Chir.*, 32: 616–625, 1885.
21. HARTMANN, H. Travaux de chirurgie. Vol. 7. L'estomac et le duodénum. Paris, 1928. Masson et Cie.
22. HEMMETER, J. C. Diseases of the Stomach. Philadelphia, 1902. Blakiston's Sons.
23. HOFFMEISTER. Beitrag zur Magenchirurgie. *Beitr. z. klin. Chir.*, 59: 551, 1908.
24. HORSLEY, J. S. Surgery of Stomach and Duodenum. St. Louis, 1933. C. V. Mosby Co.
25. KIRSCHNER, M. Operative Surgery. Philadelphia, 1933. J. B. Lippincott Co.
26. KOCHER, TH. Zur Magenchirurgie bei Carcinom und bei Ulcus simplex. *Cor.-Bl. f. schweiz. Aerzte*, 28: 610–623, 1898.
27. KOCHER, TH. Zur Technik und zu den Erfolgen der Magenresektion. *Cor.-Bl. f. schweiz. Aerzte*, 23: 682, 1893.
28. KRÖNLEIN. Vorstellung einer Patienten bei welcher wegen Carcinoma Pylori die Resection Pylori mit Erfolg. ausgefuehrt worden ist. *Cor.-Bl. f. schweiz. Aerzte*, 18: 316, 1888.
29. LIVINGSTON, EDWARD M., and PACK, GEORGE T. End Results in the Treatment of Gastric Cancer. N. Y., 1939. P. B. Hoeber.
30. MAYO, WILLIAM J. Surgical cure of cancer of the gastro-intestinal canal. *St. Paul M. J.*, 12: 1–9, 1910.
31. MAYO, W. J. Radical operation for cancer of pyloric end of stomach. *J. A. M. A.*, 54: 1608–1612, 1910.
32. MAYO, W. J. Cancer of the stomach, its surgical cure. *Surg., Gynec. & Obst.*, 14: 115–119, 1912.
33. MAYO, W. J. Operative treatment of cancer of the stomach. *J. A. M. A.*, 61: 540, 1913.
34. MIKULICZ, J. Beitraege zur Technik der Operation des Magencarcinoms. *Verhandl. d. deutsche Gesellsch. f. Chir.*, 27: 252–260, 1898.
35. MOYNIHAN, B. Abdominal Operations. Vol. 1. Philadelphia, 1926. W. B. Saunders Co.
36. PACK, G. T. Indications for two-stage resection of carcinoma of stomach. *Clin. North America*, 13: 517–523, 1933.
37. PACK, G. T. Surgical and radiation treatment of gastric cancer. *South. M. J.*, 27: 747–754, 1934.
38. PACK, G. T., and SCHARNAGEL, I. M. Gastro-enterostomy with exclusion of in-operable cancer of the pylorus and the antrum. *J. A. M. A.*, 102: 1838–1841, 1934.
39. PACK, G. T., and SCHARNAGEL, I. M. The technique of gastric resection for car-cinoma. *Surg., Gynec. & Obst.*, 63: 189–197, 1936.
40. PÉAN, J. Diagnostic et traitement des tumeurs de l'abdomen et du bassin, Vol. 1. Paris, 1880. V. A. Delahaye et Cie.
41. PÓLYA, E. Zur Strumpfoersorgang nach Magenresektion. *Zentralbl. f. Chir.*, 38: 892–894, 1911.
42. REICHEL. *Verhandl. d. deutsche Gesellsch. f. Chir.*, 37: 211–212, 1908.
43. RHÉAUME, P. Technique chirurgicale, estomac et duodénum. Paris, 1932. Masson et Cie.
44. ROBSON, A. W. M., and MOYNIHAN, B. G. A. Diseases of the Stomach and Their Surgical Treatment. New York, 1904. William Wood & Co.
45. ROEDER, C. A. Total gastrectomy. *Ann. Surg.*, 98: 221, 1933.
46. ROUX, M. Chirurgie gastro-intestinale. *Rev. de chir.*, 13: 402–403, 1893.
47. RYDYGIER, R. Meine Erfahrungen ueber die von mir seit 1880 bis jetzt ausgefuehrten Magenoperationen. *Deutsche Ztschr. f. Chir.*, 58: 197–230, 1900–1901.
48. RYDYGIER, R. Die erste Magenresektion beim Magengeschwuer. *Berlin. klin. Wchnschr.*, 19: 39–41, 1882.
49. SCHOEMAKER, J. Some technical points in abdominal surgery. *Surg., Gynec. & Obst.*, 33: 591–596, 1921.

50. STEUDEL. Die in den letzten Jahren an der Czerny' sehen Klinik ausgefuehrten Magenoperationen und die Endresultate der frueheren Operationen. *Beitr. z. klin. Chir.*, 23: 1–88, 1899.

51. STEUDEL. Die neureren Magenoperationen in der Czerny' sehen Klinik und die bisherigen Dauerfolge. *Arch. f. klin. Chir.*, 57: 459–464, 1898.

52. TERRIER, L. F., and HARTMANN, H. Chirurgie de l'estomae. Paris, 1899. G. Steinheil.

53. WALTON, A. J. Surgery of the stomaeh and duodenum. In Nelson's New Loose-Leaf Surgery, Vol. 5. New York and London, 1932. Thomas Nelson & Sons.

EDITORIALS

IT'S WORTH SEEING

THIS is not written to boost the New York World's Fair. Many of us have been long since fed up on the publicity that seems to be a necessary part of such a gigantic venture. We ourselves, as a matter of fact, vowed that we would wait until October to visit the Fair, and then perhaps spend an hour or two there.

Somehow or other, we found ourselves there shortly after the opening—and one visit has led us there repeatedly since. The amusement areas, fireworks and water displays are impressive to be sure, but the exhibits of technical and educational value are even more to our liking. The mere creation of the fairyland of light and color upon the swamps and garbage dumps which formerly existed upon the site is a sufficient testimony to the power of man and a grandiloquent tribute to public health.

If you plan to go to the Fair, be sure to spend some time in the Medical and Public Health Exhibit. The building occupies a large ground space, is attractively and intelligently planned and architecturally pleasing. We thought it well done and in excellent taste, even before we learned that the architect was none other than the son of George Kosmak, nationally known physician and editor of *The American Journal of Obstetrics and Gynecology.*

The exhibit is, of course, planned and geared for lay consumption, but will prove interesting to any medico. We ourselves were particularly struck by the divisions on maternal welfare, cancer, tuberculosis and syphilis, but this probably reflects only the fact that these are fields of particular interest to us. The Popular Superstitions show is enthralling, and we lingered long at an exhibit describing cell division, just to hear high school and college students explaining to their less scientifically minded companions what was taking place.

Long lines of individuals are waiting at some exhibits. Over four million people have visited this building alone. These have undoubtedly come away with greater respect for scientific medicine than they had when they entered. It's marvelous propaganda—much needed in these troublesome and hectic days.

Best of all is the prospect that these carefully planned exhibits are not to be interred in rubbish heaps when the Fair closes, since the most valuable, together with other material, are to be perpetuated in an American Museum of Health to be established in New York City after the Fair itself is over. The Mayor of the city has promised to find a site and the money to support the permanent institute will be provided by philanthropic societies and insurance companies.

T. S. W.

NEXT TO VALOUR

IT is not the practice or the policy of THE AMERICAN JOURNAL OF SURGERY to comment on non-medical literature. However, we are deviating for once from the general rule to touch shortly on "Next to Valour," a novel by John Jennings, Jr., recently published by Macmillan. It is splendid writing, crammed with authentic and accurate history and enough adventure for any man.

We write this because the author is the son of John E. Jennings, member of our Editorial Board, although the novel can stand on its own merits. We were sorry when we had finished it—although it runs some eight hundred pages.   T. S. W.

ALBERT MATHIEU

1881–1939

TO meet the deep and sudden sense of loss occasioned by the death of Albert Mathieu it is difficult to bring forward any of the factors which sometimes compensate. Mathieu was too young—if not perhaps in years, certainly in spirit—too productive, too vital, too strong in the hearts of those who were fond of him. Just as there seemed too little to prepare his friends for his death, there seems too little to compensate.

Albert Mathieu was born in 1881 in Faribault, Minnesota, where his early schooling was obtained. His medical studies were pursued at the University of Minnesota. After his required period of training he took up a general practice in South Bend, Washington, later moving to Raymond, Washington. During the World War he entered the army as a first lieutenant in the medical corps and served at Camp Lewis. His intimate contacts during the war with men well advanced in specialty practice convinced him of the ultimate futility of his former type of practice. At about the age of 37 he undertook a complete graduate training in the specialty of obstetrics and gynecology at the Lying-in Hospital in New York. In 1920 he began the practice of obstetrics and gynecology in Portland, Oregon, and registered continued progress in every branch of this specialty up to the actual moment of the illness which preceded his death.

Albert Mathieu was an independent and aggressive thinker. He was willing to stand behind a well-grounded opinion quite regardless of the opinions of others. He worked and studied constantly, wrote widely and well, held office in almost every important organization in his specialty and made for himself an enduring place in the hearts of his friends and intimates. He was a warm-blooded, sometimes impetuous man. He admired men of action and accomplishment, studied them and learned from them. Weakness, inconsistency, and lack of courage he despised. He was seldom if ever in robust health, especially in his later years. Yet he was always stimulating and amusing company to those whom he liked and understood. He was generous to a fault. Not everyone who knew him was his friend; yet he leaves unquestionably the deepest sense of loss and the most unmitigated sorrow.

ALBERT W. HOLMAN, M.D.

LAXATIVE INDUCED SPREADING PERITONITIS COMPLI-CATING ACUTE PERFORATIVE APPENDICITIS*

RESULTS OBTAINED WITH THE USE OF PERFRINGENS—CLOSTRIDIUM WELCHII ANTITOXIN AND A PARTIALLY MAINTAINED GLUCOSE METABOLIC BALANCE

John O. Bower, m.d.,   John C. Burns, m.d.

PHILADELPHIA, PENNSYLVANIA

AND

Harold A. Mengle, m.d.

FRANKLIN, NORTH CAROLINA

A preliminary report based on the treatment of twenty-five cases of ileus—fourteen with acute intestinal obstruction and eleven with spreading peritonitis—by the administration of perfringens (Clostridium welchii) antitoxin was published by Bower and Clark in 1928.[1] In nine of the eleven patients with spreading peritonitis, the causative factor was acute perforative appendicitis. A review of the literature in the United States prior to this date fails to reveal reports of the administration of perfringens antitoxin for the treatment of spreading peritonitis due to a perforated appendix.

In Europe, however, Delbet,[2] Micheal,[3] Williams[4] and Weinberg[5] had reported series of patients treated with the perfringens antitoxin. While the majority of patients treated had spreading peritonitis due to a perforated appendix, Delbet and Weinberg reported good results following its use in the treatment of the unruptured gangrenous appendix. I mention this because we have recently reported the finding of the antitoxin to the Clostridium welchii in the peritoneal exudate of patients operated upon for acute appendicitis in approximately 33 per cent of instances. The

treatment of a nonperforative appendicitis by passive immunization or by drainage therefore is not indicated. In 1936, Priestly and McCormack[6] reported a series of thirty-two patients treated with perfringens antitoxin with good results.

Since the preliminary report in 1928, we have been using perfringens antitoxin in the treatment of peritonitis where anaerobes were suspected of playing a part in the etiology. Experimental investigations were conducted during the same period on several different methods of treatment; these and the results of a clinical study of spreading peritonitis have been reported.[7,8,9,10,11,12,13,14,15,16,17]

The main purpose of this report is to place before surgeons the immunologic aspect of the management of spreading peritonitis which had not been emphasized heretofore. While passive immunization may be considered the feature of this article, we realize that other factors, some of great importance, play a part in the outcome. These factors may be divided into two groups—those in which the surgeon has no part and those in which he plays an important rôle. In the brief abstracts which follow, reference will be made to a study

of over 18,000 records of patients operated upon for acute appendicitis and peritonitis in twenty-eight hospitals in Philadelphia. Included in this group are 2,573 patients who had spreading peritonitis; their clinical records were reviewed a second time for a comparative study with the results obtained in this group of ninety-eight cases. The large group will be considered a control group and will be referred to subsequently as such.

Factors which influence the mortality of spreading peritonitis beyond the control of the surgeon are:

Age. Spreading peritonitis causes approximately 90 per cent of the deaths of patients admitted to hospitals with a diagnosis of acute appendicitis. The average age of those that die is 27 years; in the reported group 26 years.

Delay in Hospitalization. The average time between onset of symptoms and operation in the control group was 54.51 hours; in the reported group 93 hours.

Laxatives. The mortality increases with the administration of laxatives. In the control group a definite history of laxative administration was obtained in 1,408 cases; 153 did not receive laxatives, and sixteen, or 10 per cent, died; 1,119 had received one laxative, and 250, or 22 per cent, died; 135 had received more than one laxative, and 39, or 29 per cent, died.

In the reported group fifteen had not received a laxative, and one, or 7 per cent, died; fifty-six had received one laxative, and six, or 11 per cent, died; and seventeen had received more than one laxative, and two, or 12 per cent, died. The results obtained by the treatment outlined in this communication, I believe, are responsible for the great difference in the mortality percentage of the two groups. In this series of ninety-eight patients, eighty-eight, or 90 per cent, had received laxatives—this group is called, therefore, the Laxative Induced Spreading Peritonitis Group.

Complications. The most frequent complications of spreading peritonitis accompanying perforation of an acute appendix are acute inflammatory lesions of the lung and metastatic abscesses. The incidence of the complications in the control group cannot be used for comparison because of the small percentage of patients in the held group. In our own series the incidence of major complications in the immediate operative group was 21.15 per cent; in the held group 30.45 per cent. Metastatic abscess developed more frequently in the immediate operative group and pulmonary complications in the delayed.

Morphine Preoperatively. The percentage of patients suffering with spreading peritonitis who have been given morphine before admission to the hospital is gradually diminishing. That it still occurs too often, however, is shown by the fact that in one year fifty-five physicians in Philadelphia gave morphine to that number of patients before admission to the hospital and four died.

Enemas before Admission. Enemas may interfere with the localization of a spreading process; we have observed spreading peritonitis so induced postoperatively. Prior to hospitalization they are given infrequently as compared with laxatives, but when given, the result bears a direct relation to the stage and extent of the spreading process—given early in an advanced stage, the induced peristalsis may produce an increase in absorption sufficient to cause death; given late, when the process is regressing, a metastatic abscess only may develop.

Abdominal Palpation. Repeated abdominal palpation, particularly when forceful, interferes with the localization of a spreading peritonitis and should be avoided. Examination of the abdomen to determine the degree of rigidity and distention is necessary, but it can be done gently, the palm being placed so that contact with the epidermis only is made. Flexion of the distal phalanx will enable the examiner to estimate the degree of muscle tension. By applying the stethoscope—an instrument many surgeons have discarded—lightly to the abdomen at regular intervals, an ad-

vance or regression of the process can usually be determined.

Application of Ice Bag, Hot Water Bottle or Poultice. If patients would not use and the family physician would not order, but advise against applying a hot water bottle, ice bag or poultice to the abdomen in acute abdominal pain; if surgeons alone were given the prerogative of ordering them, the mortality of all the acute lesions of the abdomen requiring surgery would be tremendously diminished. What little good they may do is more than offset by the accompanying false sense of security. The patient may, by his attitude, influence the attending physician against operative interference.

FACTORS WHICH INFLUENCE THE MORTALITY OF SPREADING PERITONITIS CONTROLLED BY THE SURGEON

Correct Diagnosis. There are comparatively few errors of diagnosis made by surgeons when the serous membrane of the appendix is intact or when a frank spreading or local peritonitis is present, but the early subacute perforation with spreading peritonitis is frequently not diagnosed before operation. This is not difficult to explain. The gross difference between a localized mass, serosa intact appendix, which may be removed without contamination of the peritoneal cavity and an early perforation which cannot be removed without additional contamination of the peritoneal cavity is approximately 100 microns, yet if a spreading peritonitis develops following an attempt at removal of the latter the mortality is 360 times greater. Surgeons will do well to check over their diagnoses of early perforation; in this series the frank spreading peritonitis was diagnosed in 85 per cent of instances, the incipient perforation in 45 per cent.

The Evaluation of the Immunologic Response. Surgeons as a rule speak of this as the resistance or the reaction of a patient to an infection. An appreciation of the difficulties encountered in evaluating the immunologic response may be obtained by the following description of two cases which illustrate the two types most frequently observed by surgeons:

CASE I. The acute inflammation of the appendix is accompanied by a high temperature and pulse, a marked increase in the neutrophiles, definite physical signs—this patient has an adequate immunologic response and is fortunate in two respects. The lesion is very likely to be diagnosed promptly and the patient operated upon before perforation occurs; if perforation does take place and he is properly managed, he will develop a high degree of immunity.

CASE II manifests a milder immunologic response; moderate rise in temperature and pulse rate, little or no increase in neutrophiles—indefinite physical signs. This patient will react satisfactorily to the removal of the serosa intact appendix, but if there is perforation, his immunologic response will be commensurate with his preoperative symptoms and signs.

The immunologic response in the first case can be definitely evaluated by the surgeon; the patient's defensive mechanism has responded adequately to a virulent micro-organismal invasion. In the second instance, the surgeon cannot be so sure of his evaluation of his patient's reaction—it may be due to virulent micro-organisms operating in tissues of low resistance or the micro-organisms may be avirulent and the patient's response adequate. To summarize —these two cases, the prototypes of over 90 per cent of patients admitted with acute appendicitis, show that a consideration of the immunologic response is necessary only when the serosa is ruptured. In the unruptured, only one in 183 dies; in the ruptured, one in eight, or a difference of 2,200 per cent.

When to Operate. As previously stated, the immunologic aspect of the management is emphasized in this communication. It requires time for the development of either local or general immunity. This is just as true of spreading peritonitis as it is of a cellulitis, in the management of which surgeons have adopted the watchful wait-

ing principle. Gynecologists have applied this knowledge to the management of pelvic peritonitis.

Surgeons have been divided in their opinions regarding the value of time, the status of immediate or delayed operation, in the management of spreading peritonitis due to a perforated appendix mainly because immunologists have failed to submit conclusive evidence supporting either one or the other opinion. Recent investigations on the bacteriologic,[7] the immunologic,[8] and the clinical aspects,[9] of the problem seem to indicate that the factor, *Time,* is second in importance only to what is done at operation in the management of these patients.

Type of Anesthesia. The surgeon's responsibility to the patient includes the selection of the anesthetist as well as the kind of anesthetic to be administered. This is not difficult when operating for acute appendicitis. Patients with appendicial spreading peritonitis, however, frequently develop major complications and the type of anesthetic and its administration may be contributing factors in the development of the two most frequent, pneumonitis and metastatic abscesses.

Ether is still the safest general anesthetic, but Mengle[18] has shown that in the earliest stages of a spreading peritonitis, the respiratory excursions accompanying its administration increase the rapidity of peritoneal absorption. In addition, the movements of the diaphragm, the diminished relaxation of the abdominal muscles and the frequency with which distended loops of intestine crowd into the operative field, make the locating and quarantining of the infected area more difficult. This entails more time hence more anesthetic; these, together with the trauma to the endothelial cells because of the increased manipulation, result in an increase of absorption of toxic products which cannot always be successfully combated by the patient.

Local novocaine was used most frequently in this series because the initial operation in the majority of patients consisted of an incision and the insertion of a single drain. Spinal anesthesia was the second choice. The relaxation of the abdominal muscles which it alone produces, facilitates exposure of the process and apparently diminishes toxemia.

An analysis of the major complications and their relation to the kind of anesthetic administered in this group shows that spinal anesthesia in all probability did not contribute materially to the reduction of the incidence of complications involving the respiratory system. However, this small series of cases does not justify a definite statement. In my opinion if, when spinal anesthesia is used, its effect can be kept below the level of the sixth rib, it is the anesthetic of choice; the one exception being a well localized appendicial abscess which can be operated upon under local anesthesia. When the anesthesia ascends to the level of the fourth rib, the intercostal muscles are paralyzed, the excursion of the diaphragm may be interfered with, a drop in blood pressure occurs, blood stagnates in the right chest and passive pulmonary congestion occurs, presenting a condition favorable to the development of a pneumonitis.

What effect does the deferring of the operation have on the development of postoperative pulmonary complications? Of the eleven patients who died, four developed a lobar pneumonia; in two this disease was given as the cause and in two others the contributory cause of death; two were operated upon immediately; two were held; three of the four were given spinal anesthesia and one, ether.

What Is Done at Operation. Simple drainage, the search for or the removal of the appendix; the procedure depends to a great extent upon what the surgeon's past experiences have been in the management of these cases. Patients with spreading peritonitis are operated upon by two types of surgeons—those who operate routinely on all cases and those who consider the operation only a part of the management

of this condition. Scientific management is based on dose of antigen and the reactive capacity of the patient, which for the present can best be determined by studying closely the gross changes at operation and correlating them with symptoms and signs before, and what happens to the patient after operation.

What Is Done if the Patient Is Not Operated upon Immediately. The importance of a preoperative and postoperative management instituted and carried out by a group composed of the chief surgeon, associates, intern and a competent clinical pathologist who is capable of making and evaluating Schilling hemograms, cannot be overestimated. A disease which carries with it a mortality of from 25 to 40 per cent, requires constant medical supervision. It is not a one man job—a patient's chance for recovery is increased markedly with such a group in attendance. The treatment should consist of Fowler position, nothing by mouth, perfringens antitoxin or convalescent serum or both intramuscularly, glucose and saline intravenously or by hypoclysis, morphine as indicated, and mental and physical quietude, which are always indicated.

What Is Done if the Patient Has Been Operated upon Immediately. Same as above except that abdominal dressings are not changed unless absolutely necessary and drains are not disturbed usually until the seventh day.

THE ADMINISTRATION OF PERFRINGENS ANTITOXIN IN THE TREATMENT OF SPREADING PERITONITIS DUE TO A PERFORATED APPENDIX

Perfringens antitoxin was first administered in the United States in the treatment of spreading peritonitis in 1927;[1] in 1928, we began our experimental investigations as has been stated. In one of our reports, we outlined the results of the treatment of a large number of dogs in which spreading peritonitis had been induced by ligating the appendix and its mesentery and giving large doses of castor oil.[10] Our conclusions

from this experimental work were responsible for a modification of the method of administration, dose, etc.

The perfringens* used in the treatment of the series of patients previously reported and in our early experimental work was the unconcentrated serum; 50 c.c. contained 10,000 units of perfringens antitoxin. During the past two years, we have used the concentrated serum, the 20 c.c. syringe containing 10,000 units each of perfringens and vibrion septique antitoxin with an excess estimated to be 30 per cent or over.

Indication for Treatment. Patients with serosa intact appendices and those with localized abscess were not given the antitoxin; only those with spreading processes following perforation were treated. They were divided into two groups; those operated upon immediately and then given perfringens and those held, given perfringens and then operated upon. The condition of patients on admission determined the group in which they were placed—the very sick were usually held; those in which the diagnosis was uncertain in most instances were operated upon.

Method of Administration. Patients were always tested for sensitivity. In the initial series, the antitoxin was given intravenously. When we discovered that better results were obtained by using the intramuscular methods in dogs, we used it in the treatment of man. We prefer to inject the antitoxin 2 of 3 cm. below the crest of the ilium, slightly posterior to the mid-lateral line. It is advisable to select a site for injection away from the lumbar region, especially on the right side; otherwise the swelling and tenderness which may follow might make it difficult for the examiner to differentiate between tension or rigidity due to tissue reaction and that due to an intraperitoneal process. Because of the frequency with which the appendix is located retrocecally, abscesses in this region are not uncommon.

---

* Supplied by the Research Laboratories of Sharp and Dohme.

Time of Administration. Patients who were held were given the antitoxin as soon as the diagnosis was made. Those operated upon received their injection immediately upon return from the operation.

Dose. While the maximum result—lowest mortality rate in dogs—was obtained where the total amount of antitoxin given averaged 3 c.c. per kilo of body weight, it will be observed that this amount was infrequently given in the cases reported. In this series, the average dose given intramuscularly was less than 1 c.c. per kilo of body weight; the average dose per patient, whether operated upon or held, was 44.82 c.c.

Perfringens Antitoxin Combined with Whole Blood and Convalescent Serum. When we had reached what appeared to be the minimum mortality with the use of perfringens antitoxin alone, in our experimental work, we tried combining it with whole blood, but the results were approximately the same. By using convalescent serum alone, however, taken from dogs that had recovered after spreading peritonitis, we reduced the mortality below the minimum obtained by the administration of perfringens antitoxin.[10]

We then tested lyophile convalescent serum of both man and dog after it had stood as long as 444 days after lyophilization and found in both instances that it had maintained its original antitoxic titer. We have, therefore, used both fresh and lyophilized convalescent serum alone and in conjunction with perfringens antitoxin with excellent results. When the spreading process develops in a patient with low resistance as shown by a leucopenia and a marked shift of the Schilling index, we use transfusion of a small amount of citrated blood in conjunction with perfringens antitoxin or convalescent serum.

The Effect of Perfringens Antitoxin on the Toxemia of Spreading Peritonitis. None of the forty-six patients who received perfringens antitoxin as a part of the treatment for localization before operation developed delirium. However, it occurred in one instance postoperatively, in a patient who on admission had a urea nitrogen of 50 and developed uremia. In those who were operated upon and then given the antitoxin, mental irritability and delirium were not uncommon. One patient, became delirious on the fifth postoperative day, got out of bed to calm a restless fellow patient and contracted pneumonia. I feel that this catastrophe could have been averted had the patient had a sufficient amount of antitoxin. This statement is substantiated by the following experiences:

A spreading peritonitis in a male, age 44, developed following an acute perforation of a diverticulum of the sigmoid. This was localized, a colostomy was done and an abscess drained. Ochsner treatment, including intravenous glucose solution, was instituted after operation but the patient developed a delirium which persisted for three days. Anaerobic cultures of the purulent exudate showed Clostridium welchii. Following the administration of perfringens antitoxin, delirium immediately subsided.

A second patient, a female, age 56, following a Kraske operation for carcinoma of the rectum, developed an infection of the operative field with an associated toxic psychosis. She failed to respond to the intravenous injection of glucose but improved immediately after the injection of perfringens antitoxin. Clostridium welchii was isolated culturally from the purulent discharge.

Because of the high incidence of a polymicrobic flora in spreading peritonitis, the variability of the patient's immunologic response and the parenteral administration of glucose, one could not definitely ascribe the usual drop in temperature and pulse which accompanies the localization of a spreading process solely to the administration of perfringens antitoxin.

Nausea and vomiting were rare in those given perfringens before operation but not infrequent in those operated upon immediately.

The Effect of Perfringens Antitoxin on Physical Signs. Except in the frank, fulminating type of spreading peritonitis, an examination of the abdomen may not

present sufficient evidence to enable the surgeon to diagnose a spreading process definitely. The most important physical sign is *rigidity*, but even this may be absent, or it may be difficult to evaluate the degree. This is especially true in young children and in neurotic and obese individuals. When rigidity is present before the administration of perfringens antitoxin we have noted an improvement, usually beginning in the upper left quadrant, twenty-four hours after the injection. Rigidity is gradually replaced by increase in tension, followed by relaxation, which reaches its maximum about the seventh day of the disease. A differentiation between voluntary spasm and actual rigidity can best be determined by rectal examination. We believe that this should be done only at the initial examination of the patient and again directly preceding the operation.

At the second examination one may be better able to outline the process by having the patient assume the right lateral prone position with the knees slightly flexed. Free fluid which was present on admission is usually completely absorbed forty-eight hours after the first injection of prefringens antitoxin. Distention, if present, gradually lessens, liver dulness usually returning to normal between the fourth and fifth days of the disease. We have only rarely been concerned about treatment for distention; Wangensteen siphonage was used in less than 5 per cent of patients who were held before operation in this series.

The Effect of Perfringens Antitoxin on Peristalsis. Peristalsis is actually a physical sign, but its importance in the diagnosis and prognosis of the disease justifies a separate discussion. It is not always entirely absent in a laxative-induced spreading peritonitis secondary to a ruptured appendix. We occasionally hear sounds of moderate degree in the left lower and upper quadrants and faint tinkles in the right lower abdomen. While its presence may be confusing, its absence is almost always significant of a spreading process. Peristaltic sounds heard immediately or within a

few minutes of the swallowing of fluid may at times be misleading. The duodenum, jejunum and upper ileum may contract normally, the sounds being heard clearly in the upper right abdomen immediately after taking fluid, when localization of a spreading process is occurring in the right lower quadrant. Peristaltic sounds are seldom heard in the localization region, however.

When peristalsis has been absent and perfringens antitoxin has been administered, we have consistently heard the first sounds presaging the return of normal intestinal contraction in the upper left quadrant twenty-four hours after the injection. Within twelve to twenty-four hours thereafter, there is a return of peristaltic sounds in the lower left or upper right abdomen, depending in which direction the initial spread of the infection was greatest. It is difficult at times, particularly in children, to be sure that peristalsis is not present and it is more difficult to interpret any sounds that may be heard. The initial contractions of the ileum or perhaps the jejunum, may be likened to a tinkle. As the intestinal tone returns, the sound develops more volume and frequently culminates in a miniature explosion.

We are convinced that perfringens antitoxin promotes an early return of intestinal tone. We have repeatedly observed patients who, following its administration, have had bowel movements on the third day, and frequently diarrhea. In this series, 38 per cent of the patients had a spontaneous bowel movement on an average of eighty-four hours after the first injection of perfringens antitoxin. Diarrhea was present in four cases. It may be that the reaction of the mucosa of the intestine to the antitoxin is similar to the reaction of the epidermis when an urticaria develops; the resulting congestive edema involving the mucosa may stimulate peristalsis and cause the spontaneous evacuation.

An Attempt to Maintain a Glucose Metabolic Balance. The ninety-eight patients in this group were admitted to the

hospital on an average of ninety-three hours after onset of symptoms. Each patient was restless and irritable unless he had been given morphine; the temperature and pulse had increased but had not reached a maximum; the tongue and skin were dry and the abdomen was moderately distended. During the ninety-three hours preceding admission, the patient could not retain fluids. He was seriously ill but was in what might be called "the compensatory stage," "compensating" because the liver was supplying the circulating cells with nourishment in the form of glycogen. How long can the liver do this adequately? When does the "decompensation" stage begin? There is no recognized laboratory test that can determine this. There are no definite premonitory symptoms and signs.

There is, however, one function of the liver, which if utilized to the utmost during the compensatory stage, will avert disaster. It has been proved experimentally that the liver of both man and dog can metabolize approximately 1 Gm. of glucose per kilo of body weight per hour. The average weight of the patients operated upon in this series was 55 kilos. The normal liver, therefore, could dispose of 1,320 Gm. of glucose in twenty-four hours, the equivalent of 5,280 calories. If the average liver can supply the body with this number of heat units, it is reasonable to suppose that there are times when it may be called upon to do so.

In the "compensatory" stage of spreading peritonitis, therefore the temperature is elevated; metabolism is increased; consumption of glycogen is increased over the normal. How can we supply the liver with this necessary amount of fuel? *A continuous intravenous injection* of 5 per cent glucose solution, administered thirty drops to the minute, will supply 2,700 c.c. of fluid in twenty-four hours. This amount of fluid is sufficient to maintain water balance but will supply only 135 Gm. of glucose, which when metabolized, amounts to approximately 540 calories. An attempt should be made to maintain metabolic as well as water balance.

If a patient in the "immunity developing stage" of spreading peritonitis reacts satisfactorily when given 1/10 of *what the liver can dispose of in the form of glucose, then he should react very much more satisfactorily with 1/5 to 1/3 of what the liver can dispose of.* A continuous intravenous injection of 10 per cent glucose solution, administered thirty drops to the minute, will supply a patient with 270 grams of glucose in twenty-four hours or the equivalent of 1,080 calories. We use this routinely during the early stages of the disease, diminishing the percentage of glucose when sugar appears in the urine.

SUMMARY AND CONCLUSIONS

1. The results of the management of ninety-eight patients in whom spreading peritonitis developed as a complication of acute perforative appendicitis are presented.

2. Ninety per cent of these patients received one or more laxatives. For this reason, the term "laxative-induced spreading peritonitis" is used.

3. Laxative-induced spreading peritonitis is more virulent than spreading peritonitis not so induced. Laxatives, their dose, number and kind, influence mortality.

4. The factors influencing the mortality of a spreading peritonitis are discussed in order of their importance.

5. The results of the administration of perfringens antitoxin in the treatment of laxative-induced spreading peritonitis are outlined.

6. The importance of a more complete glucose metabolic balance in the treatment of laxative-induced spreading peritonitis is emphasized.

7. The results in a group of patients suffering from spreading peritonitis who were operated upon immediately are compared with those in whom operation was deferred.

Two tables are appended showing the comparative results obtained in the management of patients with spreading peritonitis due to a perforated appendix by

operating immediately and by delaying operation until a local and a general immunity had developed.

The management of the two groups was identical with the single exception of the time at which operation was performed.

TABLE I

LAXATIVE INDUCED SPREADING PERITONITIS
COMPLICATING ACUTE PERFORATIVE APPENDICITIS
Treatment—Perfringens Antitoxin—Glucose
Metabolic Balance

	No. of Cases	No. of Deaths	Average Age	Time between Onset and Operation (Hours)	Average Dose of Perfringens, C.C.	Per Cent Mortality
Immediate operation..	52	8	27	54.60	44.73	15.30
Delayed operation	46	3	22.11	196.10	44.91	6.52
	98	11				

TABLE II

LAXATIVE INDUCED SPREADING PERITONITIS
COMPLICATING ACUTE PERFORATIVE APPENDICITIS

	No. of Cases (98)	Appendix Removed			Complications	
		No. of Cases	No. of Deaths	Per Cent Mortality	Incidence of Major Comtalications	Per Cent Mortality
Immediate operation..........	52	8	3	37.5	21.15	15.39
Delayed operation	46	2	0	0	30.43	6.52

REFERENCES

1. Bower, J. O., and Clark, J. H. Bacillus welchii (perfringens) antitoxin—its therapeutic value. *Am. J. M. Sc.*, 176: 97, 1928.
2. Delbet, P. Vaccinothérapie des poradénites. *Bull. Acad. de Méd.*, 97: 453–457 (April 5) 1927.
3. Micheal D. G. *Congress i. Internat. de Chir.*, 6: 272, 1923.
4. Williams, B. W. *Brit. J. Surg.*, 14: 295, 1927.
5. Weinberg, M., Prévôt, A. R., Davesne, J. and Renard, C. Recherches sur la bactériologie et le sérothérapic des appendicites aigues. *Ann. inst. Past.*, 42: 1167–1241, 1928.
6. Priestley, J. T., and McCormack, C. J. *Surg., Gynec. & Obst.*, 63: 675, 1928.
7. Bower, J. O., Mengle, H. A., and Paxson, N. F. The bacteriology of spreading peritonitis complicating acute perforative appendicitis. A clinical and experimental study. *Surgery*, 3: 645, 1938.
8. Bower, J. O., Mengle, H. A., and Paxson, N. F. The demonstration of antitoxin for Clostridium welchii in the blood serum of patients and dogs that have recovered from spreading peritonitis complicating acute perforative appendicitis. *J. Immunology*, 34: 185, 1938.
9. Bower, J. O. Clinical pathological classification of acute appendicitis and peritonitis complicating acute perforative appendicitis. *Am. J. Surg.*, July, 1939.
10. Bower, J. O., Burns, J. C., and Mengle, H. A. Induced spreading peritonitis complicating acute perforative appendicitis. *Surg., Gynec. & Obst.*, 66: 947, 1938.
11. Bower, J. O. Pus, the friendly enemy. *Surg., Gynec. & Obst.*, 67: 124, 1938.
12. Bower, J. O., Burns, J. C., and Mengle, H. A. Spreading peritonitis complicating acute perforative appendicitis. Experimental studies. *Arch. Surg.*, 37: 751, 1938.
13. Bower, J. O., Spreading peritonitis complicating acute perforative appendicitis. Routine operation versus scientific management. *J. A. M. A.*, 112: 11, 1939.
14. Bower, J. O., Burns, J. C., and Mengle, H. A. Prontosil and the treatment of spreading peritonitis in dogs. *J. Lab. & Clin. Med.*, 24: 240, 1938.
15. Bower, J. O. When is an acute abdomen not an acute surgical abdomen? *Pennsylvania State M. J.*, June, 1938.
16. Bower, J. O. The lucid interval and acute appendicitis. *Am. J. M. Sc.*, 195: 529, 1938.
17. Bower, J. O. The nursing of patients suffering from spreading peritonitis. *Am. J. Nursing*, January, 1937.
18. Mengle, H. A. The effect of anesthetics on lymphatic absorption from the peritoneal cavity in peritonitis. An experimental study. *Arch. Surg.*, 34: 839, 1937.

ABDOMINAL AUSCULTATION*

Roger T. Vaughan, M.D. AND Phil Thorek, M.D.

Associate Professor of Surgery, Rush Medical College; Attending Surgeon, Cook County Hospital

Assistant in Anatomy and Surgery, University of Illinois School of Medicine; Associate Surgeon, Cook County Hospital

CHICAGO, ILLINOIS

THE literature of abdominal auscultation is relatively barren. We note auscultation referred to occasionally in articles dealing with abdominal problems, but its importance is stressed mainly in the English literature. No complete, concise report has appeared recently which emphasizes its value, interpretation and technique. The information it yields warrants careful study by internist and surgeon, especially in the differential diagnosis of the acute abdomen where it plays its most important rôle. We wish to record some clinical information which the stethoscope has yielded and advocate its addition to the armamentarium of the abdominal diagnostician. It is not our intention to explain the mode of production of every single sound. Such work is in progress at the present time in physiologic laboratories. It is well to recall, in this connection, the excellent work done by Dr. Charles B. Puestow on intestinal motility. This essay, however, deals solely with the auscultatory phenomena.

To understand fully and to interpret the findings we must briefly review the accepted physiology of the gastrointestinal tract. The term "peristalsis" is used in a general way to designate the wave of muscular contraction in any hollow viscus. In a restricted sense the term may apply to an intestinal movement which consists of a wave or muscular contraction preceded by a wave of relaxation. We refer to this progressive phenomenon by such terms as "the law of the intestine" or "myenteric reflex." Cannon has subdivided peristaltic movements into the following:

(a) *Diastalsis:* a downward wave of contraction preceded by a wave of inhibition which is manifested chiefly by the small intestine. Sometimes these waves travel rapidly for a length of 2 or 3 feet and we then refer to them as "peristaltic rushes."

(b) *Catastalsis:* a downward wave of contraction *not* preceded by a wave of inhibition and manifested chiefly by the stomach.

(c) *Anastalsis:* an upward moving wave of contraction not preceded by a wave of inhibition (reverse or antiperistalsis). This reverse action is normally manifested by the proximal colon but may also appear as an abnormal manifestation or any portion of the alimentary tract.

There are some movements of the intestinal tract that are not strictly peristaltic in nature. For our purpose, we shall mention a few of these:

Rhythmic segmentation—a localized rhythmical contraction found in the small intestine which churns and subdivides the masses of food within the intestinal lumen.

Pendulum movements—which manifest themselves by a to and fro movement of the small intestine.

Haustral movements—local contractions of the colonic haustra, analogous to the rhythmic segmentation of the small intestine.

Carey has made some interesting observations on the anatomy, and histology of the bowel musculature. He disagrees with the current concept that the muscle wall consists of an inner circular and an outer longitudinal layer. By interesting experiments, he rather conclusively shows

that instead of an inner circular layer there is really a closely coiled oblique layer. He further shows that instead of an outer longitudinal layer of muscle we are dealing with another oblique layer, the coiling of which is more elongated. He further states the inner layer makes one complete turn around the intestinal lumen within a distance of every 5 mm. while it takes 15 mm. for the outer layer to make one complete turn.

Visualization of the result of the contraction of these two spiral muscles, makes it logical that the cephalad constriction is caused by a contraction of the inner layer while the caudal dilatation is produced by a contraction of the outer oblique layer. We believe that the sounds produced are a combination of two factors, (1) the contracting muscle causing (2) the onward passage of intestinal contents within the lumen of the gut.

As to innervation, peristalsis is supposedly under the control of the extrinsic and intrinsic nerves. The extrinsic control is via the sympathetic (splanchnic) nerves which inhibit, and the parasympathetic (vagus) fibers which augment peristaltic action. The intrinsic mechanism is provided by the Meissner and Auerbach plexuses.

When one listens to an abdomen for the first time he may be baffled by the number and types of sounds heard, but such variations are to be expected in view of the varieties of peristaltic movements. With a little practice normal sounds can readily be differentiated from the abnormal, not only as to quality but also as to intensity and frequency. To describe sound in words is difficult, so that we must use rather commonplace descriptive terms and convey our auditory perceptions by comparing them with analogous sounds from other sources.

Normal peristaltic sounds may be described as resembling gurgles, rustlings or clicks which tend to be continuous or even overlapping. They are usually more continuous than the heart sounds and relatively homogeneous. When slowed to the respiratory rate they are said to be def-

initely diminished and, when several respirations pass without a single peristaltic sound being heard, they are considered greatly diminished. They are normally not heard without the use of the stethoscope. Conditions which slow peristalsis tend to make the sounds fainter; louder sounds usually accompany an increased peristaltic rate. The exceptions to this rule will be discussed subsequently.

Normal variations occur in both rate and tonal intensity. The intake of food, drink and certain drugs stimulates peristalsis and increases sounds. Starvation, repeated vomiting and depressant drugs have the reverse action. Among such drugs are ether, chloroform and alcohol.

Air-swallowing and excessive colonic irritation may produce booming or explosive noises audible without a stethoscope and even at some distance; but they are not ringing or resonant like obstructional borborygmi.

Sound variations in pathologic conditions group themselves into three similar categories, namely, diminished, increased and abnormal sounds.

Complete intestinal paralysis produces complete peristaltic silence. The late Dr. J. B. Murphy used to refer to the abdomen in acute diffuse peritonitis as being "silent as the grave." Perforated peptic ulcer, diffuse appendicial peritonitis, abdominal gunshot wounds, acute pancreatitis and various acute peritonitides belong in this category. Large abdominal acute effusions of blood, bile and urine produce peristaltic silence; smaller ones at least a relative quiet.

In intestinal paresis, i.e., incomplete paralysis, the abdomen is quiet but not silent; e.g. in lesser effusions of blood, bile and urine, in the serous peritonitis or "wet belly" or acute appendicitis preceding perforation; in the acute serous or sero-hemorrhagic effusion of twisted pedicles, in the lesser hemorrhages of ruptured ectopic pregnancy, bleeding corpus hemorrhagicum or corpus luteum and abdominal injuries with some hemorrhage but no peritonitis.

A ruptured Graafian follicle diminishes peristalsis only mildly as a rule.

After general anesthesia the peristaltic sounds gradually return to something like normal in from twenty-four to forty-eight hours, the time of recovery being further prolonged by postoperative atony, abdominal hemorrhage and, most of all, by peritonitis. Neisserian peritonitis produces more often a quiet than a silent abdomen.

The peristaltic sounds are increased in incomplete mechanical ileus, spinal anesthesia, active catharsis and intraintestinal hemorrhage, such as in bleeding peptic ulcer and, probably, ruptured esophageal varix. In incomplete mechanical ileus the increased sounds are due to hypertrophied musculature of the bowel wall. In spinal anesthesia the sympathetic nerves are believed to be anesthetized, permitting the parasympathetic augmenters to act unopposed. In intraintestinal hemorrhage, blood within the intestinal lumen stimulates peristalsis in the same manner as food. In bleeding peptic ulcer the stethoscope becomes an important diagnostic aid in differentiating intraintestinal bleeding from intraperitoneal bleeding. In the former peristalsis is increased; in the latter peristalsis is greatly diminished or absent.

Obstructional borborygmi are peristaltic sounds, metallic and tinkling, bubbling or loud and resonating in character, and are pathognomonic of incomplete mechanical ileus. They are heard in no other condition. The metallic tinkling borborygmi must be differentiated from similar, metallic, splashing sounds in complete ileus which are passively produced by the palpating hand, stethoscope or diaphragmatic movement. The resonating or ringing borborygmi are produced by the peristaltic movement of distended intestinal segment lying proximal to the seat of obstruction and containing both gas and. liquid Normally, there is little or no gas in the small intestine. The presence of this metallic tinkling type of sound, therefore, signifies some variety of ileus of relatively high degree.

Among other abdominal sounds are: transmitted cardiac and respiratory sounds, splenic and liver friction rubs, the splashing sounds of pneumohydroperitoneum, coin sound, the bruit of abdominal aneurysm, crepitation from subcutaneous emphysema and the so-called mythical (?) "bruit de collision" of large gallstones knocking against one another.

Cardiac and respiratory sounds are not transmitted through the normal abdomen, but they are heard throughout the abdomen in intestinal distension. In the otherwise silent abdomen of complete paralytic ileus, the cardiac and respiratory sounds are usually strikingly prominent, being the only sounds heard except for the occasional adventitious splashes or tinkles previously mentioned.

A splenic or hepatic rub is usually due to the presence of fibroplastic exudate between visceral and parietal peritoneum and indicates a fibrinous peritonitis. A pleuritic rub is ruled out by noting the absence of breath sounds.

In a case of phlegmonous gastritis a to and fro friction rub was heard just above and to the left of the navel (abdomen shaved) where the stomach comes in contact with the anterior abdominal wall.

The splashing sound of pneumohydroperitoneum has not been described previously so far as we can determine. We have heard it only once in a case of perforated carcinoma of the stomach which showed a large pneumoperitoneum and a long fluid level in the free peritoneal cavity. We heard it first when the patient hiccuped. The sign may be elicited too, by having the patient inhale deeply and then forcefully and rapidly exhale. It is a characteristic tinkling splash like that in hydropneumothorax.

Coin sound in pneumoperitoneum is transmitted as characteristically as in pneumothorax and more loudly than in tympanites. Together with auscultatory percussion and the rubbing or scratching sound tests, this can outline the gas bubble somewhat more accurately than percussion alone.

In ascites the transmission of a fluid wave on percussion is accompanied by a "double tap" sound which is not generally known. The "double tap" is not transmitted by a fat abdominal wall as is the percussion wave at times. The tone character of the "double tap" is about like that of the so-called "split second sound" of the heart. The two taps come very close together and are due to the fact that the percussion sound wave travels through ascites a little faster than the fluid wave. Two other auscultatory tests—the "scratch and rub tests" produce a sound which is easily transmitted through meteorism and pneumoperitoneum but not through solid or cystic tumor, fluid or through the normal abdomen unless very slender.

To illustrate, we shall describe the sounds in some typical abdominal lesions. In unruptured acute appendicitis, during the first twenty-four to thirty-six hours, peristalsis is usually normal or even mildly increased; but as a serous peritoneal effusion ("serous peritonitis" or "wet belly") appears, the sounds diminish in strength and frequency and, "the wetter, the quieter." When perforating, purulent spreading peritonitis sets in, peristalsis rapidly disappears, the abdomen becomes silent (paralytic ileus) and tympanites develops in the silent, tender belly. As the appendicial inflammation becomes walled off and an inflammatory mass or "abscess" is formed, mechanical ileus due to adhesions, kinks, and inflammatory edema of the small bowel may result. In the lesser degrees of obstruction, there are resonant bubblings and gurglings in "rushes" and in the higher degrees, metallic tinklings and clicks with long silent intervals. If the abscess disappears or is evacuated, the sounds gradually return to normal as the intestines free themselves.

After a celiotomy, the abdomen is usually silent (traumatic paresis?). Intestinal sounds normally begin to return within the first twenty-four to forty-eight hours after operation. If the abdomen remains silent longer, we may suspect some intraperitoneal complication such as peritonitis or hemorrhage.

Infections of the abdominal wall do not alter peristaltic sounds except by spreading to the peritoneal cavity.

Perforated peptic ulcer presents probably immediately (reflex?) a silent abdomen of the quietest type. In the forme fruste type of ulcer perforation, peristaltic sounds begin to return soon after leakage stops and they strengthen as peritonitis subsides.

The sounds are loudest with chronic incomplete mechanical ileus. Here the bowel may become excessively hypertrophied and the sounds so loud as to be audible without the stethoscope. As the obstruction becomes more complete, the "rushes" and other sounds become fewer and metallic "tinklings" make their appearances until they too become blotted out in the silence of complete obstruction; in brief, "the tighter the obstruction, the quieter, the abdomen." The silent obstruction brooks no delay, if it is not already seen too late. The noisy obstruction may have a food-obturation factor removable by oil and flushings. It can also be a gangrenous Richter hernia.

Acute salpingitis produces the least silent acute perforative peritonitis, perhaps because the sigmoid, ileum and omentum wall off the Neisserian infection in the pelvis and prevent its upward spread, so that most of the intestine continues to function and produce sounds. Here, too, kinking of the ileum may result in mechanical obstruction and obstructive borborygmi be the guide to its diagnosis.

In extra-abdominal lesions, such as pericarditis with effusion, coronary thrombosis, angina pectoris, pneumonia, dissecting aneurysms, diaphragmatic pleurisy and acute osteomyelitis, the peristaltic sounds remain normal unless diminished by fever, abstinence from food or other accompanying conditions. Neither does renal colic, pyelitis, acute unruptured cholecystitis materially affect peristaltic sounds per se.

In the traumatic abdomen the persistence or early return of normal sounds is a

A ruptured Graafian follicle diminishes peristalsis only mildly as a rule.

After general anesthesia the peristaltic sounds gradually return to something like normal in from twenty-four to forty-eight hours, the time of recovery being further prolonged by postoperative atony, abdominal hemorrhage and, most of all, by peritonitis. Neisserian peritonitis produces more often a quiet than a silent abdomen.

The peristaltic sounds are increased in incomplete mechanical ileus, spinal anesthesia, active catharsis and intraintestinal hemorrhage, such as in bleeding peptic ulcer and, probably, ruptured esophageal varix. In incomplete mechanical ileus the increased sounds are due to hypertrophied musculature of the bowel wall. In spinal anesthesia the sympathetic nerves are believed to be anesthetized, permitting the parasympathetic augmenters to act unopposed. In intraintestinal hemorrhage, blood within the intestinal lumen stimulates peristalsis in the same manner as food. In bleeding peptic ulcer the stethoscope becomes an important diagnostic aid in differentiating intraintestinal bleeding from intraperitoneal bleeding. In the former peristalsis is increased; in the latter peristalsis is greatly diminished or absent.

Obstructional borborygmi are peristaltic sounds, metallic and tinkling, bubbling or loud and resonating in character, and are pathognomonic of incomplete mechanical ileus. They are heard in no other condition. The metallic tinkling borborygmi must be differentiated from similar, metallic, splashing sounds in complete ileus which are passively produced by the palpating hand, stethoscope or diaphragmatic movement. The resonating or ringing borborygmi are produced by the peristaltic movement of distended intestinal segment lying proximal to the seat of obstruction and containing both gas and. liquid Normally, there is little or no gas in the small intestine. The presence of this metallic tinkling type of sound, therefore, signifies some variety of ileus of relatively high degree.

Among other abdominal sounds are: transmitted cardiac and respiratory sounds, splenic and liver friction rubs, the splashing sounds of pneumohydroperitoneum, coin sound, the bruit of abdominal aneurysm, crepitation from subcutaneous emphysema and the so-called mythical (?) "bruit de collision" of large gallstones knocking against one another.

Cardiac and respiratory sounds are not transmitted through the normal abdomen, but they are heard throughout the abdomen in intestinal distension. In the otherwise silent abdomen of complete paralytic ileus, the cardiac and respiratory sounds are usually strikingly prominent, being the only sounds heard except for the occasional adventitious splashes or tinkles previously mentioned.

A splenic or hepatic rub is usually due to the presence of fibroplastic exudate between visceral and parietal peritoneum and indicates a fibrinous peritonitis. A pleuritic rub is ruled out by noting the absence of breath sounds.

In a case of phlegmonous gastritis a to and fro friction rub was heard just above and to the left of the navel (abdomen shaved) where the stomach comes in contact with the anterior abdominal wall.

The splashing sound of pneumohydroperitoneum has not been described previously so far as we can determine. We have heard it only once in a case of perforated carcinoma of the stomach which showed a large pneumoperitoneum and a long fluid level in the free peritoneal cavity. We heard it first when the patient hiccuped. The sign may be elicited too, by having the patient inhale deeply and then forcefully and rapidly exhale. It is a characteristic tinkling splash like that in hydropneumothorax.

Coin sound in pneumoperitoneum is transmitted as characteristically as in pneumothorax and more loudly than in tympanites. Together with auscultatory percussion and the rubbing or scratching sound tests, this can outline the gas bubble somewhat more accurately than percussion alone.

In ascites the transmission of a fluid wave on percussion is accompanied by a "double tap" sound which is not generally known. The "double tap" is not transmitted by a fat abdominal wall as is the percussion wave at times. The tone character of the "double tap" is about like that of the so-called "split second sound" of the heart. The two taps come very close together and are due to the fact that the percussion sound wave travels through ascites a little faster than the fluid wave. Two other auscultatory tests—the "scratch and rub tests" produce a sound which is easily transmitted through meteorism and pneumoperitoneum but not through solid or cystic tumor, fluid or through the normal abdomen unless very slender.

To illustrate, we shall describe the sounds in some typical abdominal lesions. In unruptured acute appendicitis, during the first twenty-four to thirty-six hours, peristalsis is usually normal or even mildly increased; but as a serous peritoneal effusion ("serous peritonitis" or "wet belly") appears, the sounds diminish in strength and frequency and, "the wetter, the quieter." When perforating, purulent spreading peritonitis sets in, peristalsis rapidly disappears, the abdomen becomes silent (paralytic ileus) and tympanites develops in the silent, tender belly. As the appendicial inflammation becomes walled off and an inflammatory mass or "abscess" is formed, mechanical ileus due to adhesions, kinks, and inflammatory edema of the small bowel may result. In the lesser degrees of obstruction, there are resonant bubblings and gurglings in "rushes" and in the higher degrees, metallic tinklings and clicks with long silent intervals. If the abscess disappears or is evacuated, the sounds gradually return to normal as the intestines free themselves.

After a celiotomy, the abdomen is usually silent (traumatic paresis?). Intestinal sounds normally begin to return within the first twenty-four to forty-eight hours after operation. If the abdomen remains silent longer, we may suspect some intraperitoneal complication such as peritonitis or hemorrhage.

Infections of the abdominal wall do not alter peristaltic sounds except by spreading to the peritoneal cavity.

Perforated peptic ulcer presents probably immediately (reflex?) a silent abdomen of the quietest type. In the forme fruste type of ulcer perforation, peristaltic sounds begin to return soon after leakage stops and they strengthen as peritonitis subsides.

The sounds are loudest with chronic incomplete mechanical ileus. Here the bowel may become excessively hypertrophied and the sounds so loud as to be audible without the stethoscope. As the obstruction becomes more complete, the "rushes" and other sounds become fewer and metallic "tinklings" make their appearances until they too become blotted out in the silence of complete obstruction; in brief, "the tighter the obstruction, the quieter, the abdomen." The silent obstruction brooks no delay, if it is not already seen too late. The noisy obstruction may have a food-obturation factor removable by oil and flushings. It can also be a gangrenous Richter hernia.

Acute salpingitis produces the least silent acute perforative peritonitis, perhaps because the sigmoid, ileum and omentum wall off the Neisserian infection in the pelvis and prevent its upward spread, so that most of the intestine continues to function and produce sounds. Here, too, kinking of the ileum may result in mechanical obstruction and obstructive borborygmi be the guide to its diagnosis.

In extra-abdominal lesions, such as pericarditis with effusion, coronary thrombosis, angina pectoris, pneumonia, dissecting aneurysms, diaphragmatic pleurisy and acute osteomyelitis, the peristaltic sounds remain normal unless diminished by fever, abstinence from food or other accompanying conditions. Neither does renal colic, pyelitis, acute unruptured cholecystitis materially affect peristaltic sounds per se.

In the traumatic abdomen the persistence or early return of normal sounds is a

rather certain reassurance of absence of serious intraperitoneal damage. On the other hand, should the abdomen become silent and remain so, we have an indication for surgical intervention unless pain, tenderness and rigidity are absent.

In acute rupture of the bladder, if the urine is sterile, the sounds gradually disappear, while with infected urine, they disappear rapidly.

The abdomen should be auscultated both before and after attempting reduction of a hernia. If a viable segment of intestine is reduced the obstruction is completely overcome, the sounds reappear and become *increasingly normal and vigorous. If ob*structive borborygmi were present before reduction they gradually return to normal tonality following reduction. If a hernia is reduced without relieving the obstruction (reduction *en bloc* or of a gangrenous loop) the abdomen remains silent in incomplete obstruction, or borborygmi appear if obstruction remains incomplete. When an intestinal perforation follows taxis, the abdomen becomes silent instead of regaining normal sounds and becomes increasingly tender, instead of less tender, until it reaches the absolute silent stage of a well-developed peritonitis.

CONCLUSIONS

1. Abdominal auscultation deserves a place in the diagnostic aramentarium of the physician.

2. Normal and abnormal sounds have been discussed and described.

3. The characteristic silence of spreading peritonitis is emphasized.

4. The significance of obstructive borborygmi is outlined.

REFERENCES

BLUMER, GEORGE. Bedside Diagnosis. Philadelphia, 1938. W. B. Saunders Co.

BOAS, I. L. Diagnostik und Therapie der Magenkrankhciten, p. 70. Leipzig, 1925. G. Thieme.

CANNON, W. B. Auscultation of rhythmic sounds produced by stomach and intestines. *Am. J. Physiol.*, 14: 339–353, 1905.

CAREY, E. J. Studies in structure and function of small intestine. *Anat. Rec.*, 21: 189–215, 1921.

CHARBONNIER, A. L'auscultation dans les affections chirurgicalcs aigues de l'abdomen. *Rec. méd. de la Suisse rom.*, 56: 513–585, 1936.

DA COSTA, J. C. Physical Diagnosis, 4 Ed., Philadelphia, 1919. W. B. Saunders Co.

DUNNER, L., and NEUMANN, R. Perkussion, Auskultation und Palpation der Brust und Bauchorganc. Vienna, 1924. Urban u. Schwarzenburg.

EWART, W. A. Note on auscultatory friction as an adjunct to auscultatory percussion in abdominal exploration. *Lancet*, 2: 551, 1898.

FLINT, A. Manual of Auscultation and Percussion. Philadelphia, 1912. Lea & Febiger.

LIDSKY, A. T. Auscultation of abdominal cavity in diagnosis of acute surgical diseases. *Vestnik kbit.*, 36: 56–57, 1934.

PUESTOW, C. B. Studies on activity of isolated intestina segments. *Proc. Staff Meet. Mayo Clin.*, 6: 131 (March 4) 1931.

PUESTOW, C. B. Simultaneous electrograms and myograms from isolated intestinal segments in unanaesthctized dogs. *Proc. Soc. Expec. Biol. & Med.*, 29: 901 (April) 1932.

PUESTOW, C. B. Activity of isolated intestinal segments. *Arcb. Surg.*, 24: 545 (April) 1932.

PUESTOW, C. B. Action of ccrtain drugs on isolated intestinal transplants. *Proc. Soc. Exper. Biol. & Med.*, 30: 389, 1933.

PUESTOW, C. B. Studics on origin of autonaticity of intestines; action of certain drugs on isolated intestinal transplants. *Am. J. Pbysiol.*, 106: 682 (Dec.) 1933.

PUESTOW, C. B. Postoperative care. *J. Indiana Stale M. A.*, 30: 338–340, 1937.

REHFUSS, M. E. Diagnosis and Treatment of Diseases of the Stomach. Philadelphia, 1927. W. B. Saunders Co.

STEVENS, N. C. Auscultation of abdomen; aid to diagnosis. *New England J. Med.*, 211: 108–110, 1934.

STEVENS, N. C. Auscultation of the abdomcn. *New England J. Med.*, 215: 22–26 (July 2) 1936.

WATKINS, T. J. Abdominal palpation and auscultatory percussion. *Surg., Gynec. & Obst.*, 11: 426–429, 1910.

WANGENSTEEN, O. H. Christopher's Textbook of Surgery, p. 1201. Philadelphia, 1936. W. B. Saundcrs.

THE OPERATIVE TREATMENT OF SCOLIOSIS*

Henry F. Ullrich, m.d., sc.d. (med.)

BALTIMORE, MARYLAND

THE objective in the treatment of scoliosis should be completely to correct the curvatures and maintain correction. That many curvatures can be completely corrected cannot be denied; indeed, with some of the mild ones, or even the moderately severe ones, lateral bending alone is sufficient to do this. Others with severe deformity and accompanying trunk displacement cannot be so corrected, but they are by no means beyond help. With treatment in these cases the deformity may be lessened and the trunk displacement reduced. The problem is how to maintain the correction or the correction and compensation. This we believe is best done by operative fusion of a portion of the spine.

All patients who are known to have had poliomyelitis as well as those who have curvatures, are referred to a special division of the clinic. Such patients are given a general physical check-up. Sitting and standing heights are measured, as well as the length of the lower extremities. At the same time the list or displacement of the trunk is estimated. Then roentgenograms of the entire spine are taken both in the sitting and recumbent positions. At times one needs further roentgenographic study, which may be done later. The patient is sent to the gymnasium for exercises to improve posture, increase flexibility of the spine, and keep him in close touch with the clinic. This is often continued because of delay in securing operative permission.

On the data obtained we should be able to give a prognosis. Risser and others have observed that a curvature will increase until the growth period has ceased, and after that there will be no further increase in the deformity. This has been found to be true for all curves, regardless of etiology. At this point one must select the proper patients for treatment by fusion. Cases selected must warrant the long time spent in the hospital that is necessary for a satisfactory result.

The next problem is to estimate what result may reasonably be expected with correction, or correction and compensation, followed by operative fusion of the spine.

The patients that are suitable candidates for such treatment may be divided into the following groups:

1. Those with mild or very flexible, progressing curves that may be completely corrected. (Figs. 1–4.)

2. Those with moderate curves where partial correction is sought, as well as the reëstablishment of body balance. (Figs. 5–8.)

3. Those with severe deformity, together with marked displacement of the trunk, where appreciable correction of the primary curve is not the sole object, but rather the reëstablishment of body balance. (Figs. 9–11.)

4. Those with undue pain or fatigue, regardless of the mildness of severity of the deformity, even if the growth period has passed.

The patients in the first group are advised this treatment especially when further growth is expected because we know that to let such a curve go untreated would result in the development of structural changes in the thorax and vertebral bodies, and as time goes on the possibility of complete correction would be proportionately lessened. On the other hand, to advise such treatment on a young or immature curve where the ends are not definite and the

FIG. 1.   FIG. 2.
FIGS. 1 AND 2. Back and side views of patient in Group 1.

FIG. 3.   FIG. 4.
FIGS. 3 AND 4. Forward bending and x-ray views of same patient as in Figures 1 and 2.

FIG. 5.   FIG. 6.
FIGS. 5 AND 6. Back and side views of patient in Group 2.
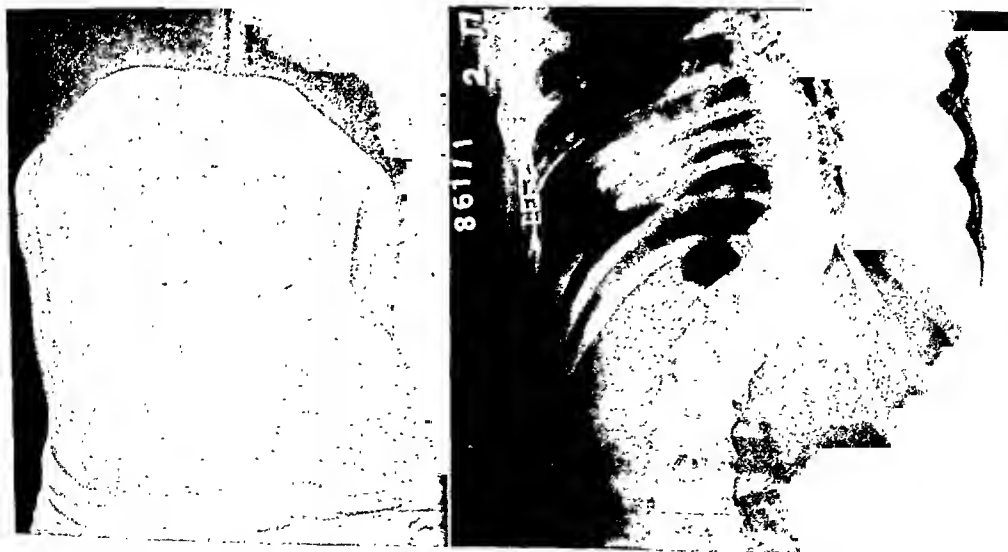

FIG. 7.   FIG. 8.
FIGS. 7 AND 8. Forward bending and x-ray views of same patient as in Figures 5 and 6.
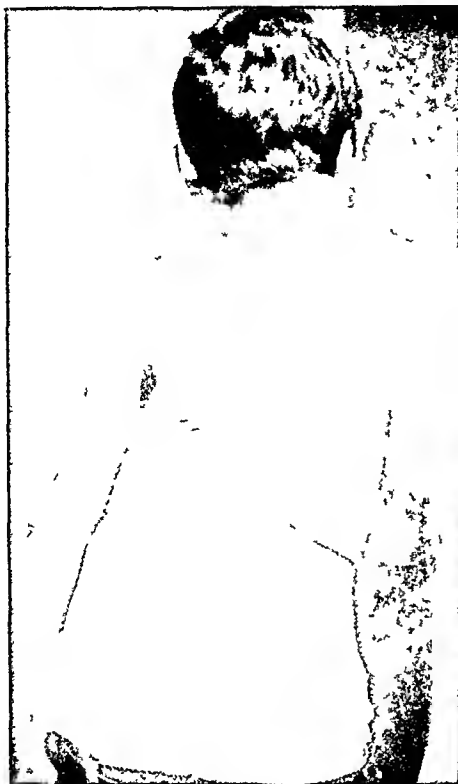
FIG. 9.   FIG. 10.

FIGS. 9 AND 10. Back and side views of patient in Group 3.

FIG. 11. Forward bending view of same patient as in Figures 9 and 10.
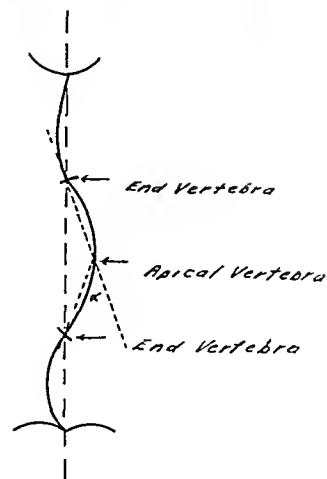
FIG. 12. Diagrammatic representation of method of curve measurement.

primary curve uncertain would be equally dangerous. To correct and fuse a compensatory curve would leave an imbalance in the

Fig. 13. Diagram of triple curve with middle curve primary.

their backs should present little or no deformity even to the trained eye.

When we see a patient with a severe rigid
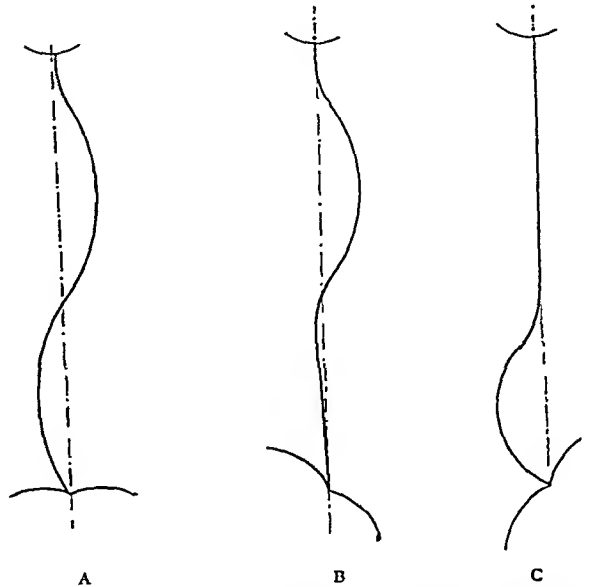
Fig. 14. A, type of curvature requiring study with pelvic tilt x-ray. B, behavior with thoracic curve primary. C, behavior with lumbar curve primary.

primary curve, and the patient would have been far better off untreated.

The patients in the second group comprise probably the largest number of candidates for operative treatment. They consist for the most part of pre-adolescents whose deformity has been recently noted at school or at home and who have been sent for advice and treatment. Some have curves in the lumbar spine that can be completely corrected, but in most, the primary curve is higher, making the prognosis for complete correction somewhat less favorable. But if a patient with moderate deformity can be corrected to a point below which the deformity is apparent, and, at the same time, whatever displacement there is be completely corrected, a very good clinical result should be expected. These patients are told beforehand that although the roentgenogram will not show a geometrically straight spine,

curvature, with its attendant structural changes in the vertebral column and thorax, as well as poor vital capacity and lowered general health, we should not give up in despair and say that since the spine cannot be appreciably straightened there is nothing to be done. These patients often have gone from clinic to clinic, and we sometimes see them suffering from mental abberation because of an inferiority complex due to the deformity. These individuals are told they will receive little or no correction of the curve itself but should obtain a reëstablishment of body balance that will greatly improve the general health and make the treatment a worthwhile process.

Often patients come in complaining of a painful area of the spine, and subsequent examination reveals the presence of scoliosis. Such patients often do not have deformities sufficiently severe to warrant a very long period of treatment. Exercises and massage are begun, and if these are not successful, fusion of the painful area is advised. Since the time in bed is not much
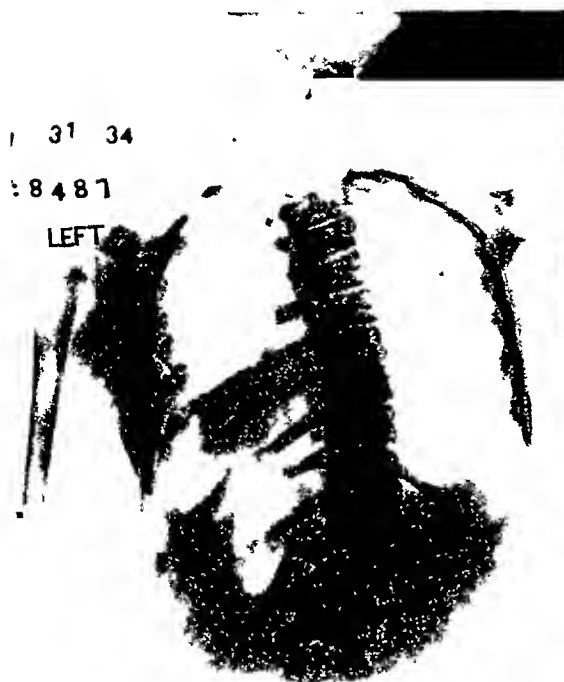
FIG. 15. A congenital curve which is partly due to a wedged fifth lumbar vertebra.

FIG. 16. Disappearance of the curve with elevation of the right side of the pelvis.
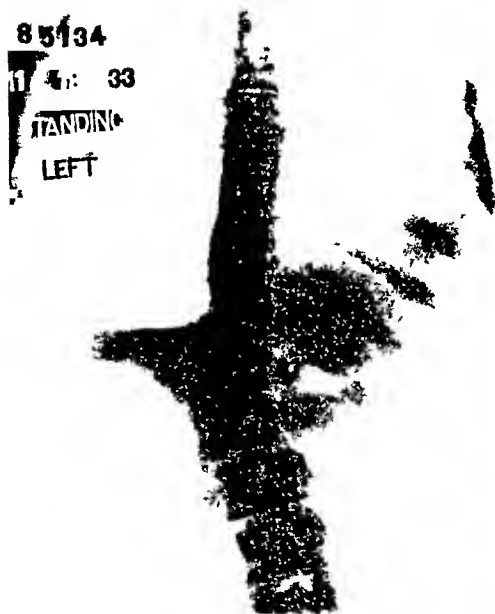
FIG. 17. Typical psoas curve taken in the sitting position.

FIG. 18. Same curve with thigh on the left side actively contracted against resistance.

longer they are put through the regular correction treatment.

Measurement of a Curve. The accurate roentgenograms in both the erect and the recumbent positions. In all subsequent measurements the same vertebrae are used.

FIG. 19. Application of wedging jacket.

FIG. 20. The wedging jacket completed.

FIG. 21. Wedging begun.

measurement of a curve is accomplished as follows; The apical vertebra and the ends or neutral vertebrae are determined. The complementary angle of lines drawn through the center of the bodies of these three vertebrae is designated as the angle of curve. This should be measured in the With reasonable care the error should not be more than 3 to 5 degrees, slightly more when kyphosis is present. (Fig. 12.)

Determination of the Primary Curve. Before one can treat a case of lateral curvature successfully it is necessary to determine the primary curve and its ex-

tent. In cases with so-called triple curves this can usually be done by inspection. (Fig. 13.) These curvatures have either a

FIG. 22. Finished jacket after correction. Ready for fenestration.

return to the erect above and below the primary curve or a compensatory one is formed above or below the primary curve. If one considers the upper curve primary it is known that the spine does not form two compensatory curves below the primary and, conversely, if one considers the lowest curve primary there are not two compensatory curves above the primary. It is known, however, that a primary curve can form two compensatory curves, one above and the other below the deforming one. Hence in this case the middle curve is the primary curve.

In cases with double curves of approximately equal extent and severity the selection of the primary curve at times offers great difficulty. (Fig. 14.) In these cases it is necessary to take roentgenograms in the sitting position, with first the right and then the left side of the pelvis elevated. This is done to study the power of the muscles acting on the sides of the convexity of the two curves. If a patient with a right dorsal, left lumbar curve be seated

with the left side of the pelvis elevated, the lumbar curve should correct for this pelvic tilt more than for the dorsal because of the

FIG. 23. Preoperative marker roentgenogram.
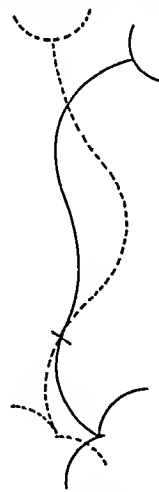
Dotted Line Represents Pelvic Tilt Film
Solid Line Represents Spine In Maximum Correction

FIG. 24 Method of superimposition of pelvic tilt film and film of spine in maximum correction.

greater flexibility of the lumbar spine. If there is no ability in the lumbar spine to correct, the weight shifts to the right in the lumbar region and must therefore be shifted to the left in the dorsal in order to center the head over the pelvis. This causes

an increase in the lumbar curve and a decrease in the dorsal, showing a weakness of the muscles to the side of the convexity of the lumbar curve. Hence, the lumbar curve in this case is primary. Conversely, if the right side of the pelvis is elevated the power in the muscles acting on the right of the dorsal spine may be studied. The curve thus showing the imbalance is designated the primary curve. Rarely are primary curves seen. This does occur at times in scoliosis due to poliomyelitis.

At times we see a curve which may be thought to be due to a tilt of the fifth lumbar vertebra on the sacrum. To prove or disprove this it is necessary only to seat the patient with the superior surface of the fifth lumbar vertebra level. If the curve disappears the cause has been established. (Figs. 15 and 16.)

Curves due to psoas imbalance possess definite characteristics and should not be easily confused with other similar curves.

(Figs. 17 and 18.) These psoas curves include only the five lumbar vertebrae and the entire curve is outside or lateral to the

FIG. 25.   FIG. 26.

FIGS. 25 AND 26. Case 1, before treatment.

center line of the pelvis. These curvatures show very little rotation, and the spine above may return to the erect or show a slight compensatory curve. If the patient is placed on his back and the thigh on the concave side of the curve is actively flexed against resistance, thus creating a pull on the psoas muscle, the roentgenogram will demonstrate an increase in the curve, and, on the other hand, if the actively contracting psoas muscle on the convex side is likewise resisted the roentgenogram will show a straightening of the curve.

The Application and Care of the Wedging Jacket. The application of a comfortable, well-fitting and efficient jacket is one of the most important steps in the successful treatment of scoliosis.

A very simple apparatus is used, consisting essentially of a wooden frame with a

FIG. 27.

FIG. 28.

FIGS. 27 AND 28. After treatment.

FIG. 29.

FIG. 30.

FIGS. 29 AND 30. Case 1 in the sitting position before and after treatment.

removable steel crossbar in the middle. At each end is a windlass. One of these windlasses is used to hold taut a web strap

The hardware consists of two flat hinges and two turnbuckle lugs. The hinges may be made from ordinary strap iron, with the

Fig. 31.
Fig. 32.
Figs. 31 and 32. Case ii before operation (April 20, 1927).

attached to the steel crossbar. This should be drawn up very tightly to minimize the sag when the patient is placed upon the frame. The windlass in the other end is used to hold the head in moderate traction during the application of the jacket.

The patient is invested with stockinette extending from above the head to below the knees. Holes are cut in the sides for the arms. The stockinette is tied in a crop over the head to prevent the hair from becoming soiled during the application of the plaster. A small hole cut over the nose allows for more comfortable breathing.

Pads of thick felt are then cut and placed to distribute pressure over bony prominences and points of pressure, such as occiput, chin, axillae, spine and about the pelvis. When thick felt is used a much smoother interior can be made if the edges are bevelled.

flat surfaces roughened and perforated by steel wire to give them a better grip in the plaster. The lugs as shown may also be made from strap iron.

The patient is then placed on the frame, lying in the web strap, with the crossbar at about the level of the seventh cervical vertebra. For orientation it has been found advisable at this point to place the roentgenogram directly on the patient's chest to lessen the possibility of bending the spine against the wrong curve and to visualize the spine better during the correction process. A sling made of two pieces of muslin bandage is placed over the head and attached to a spreader, which in turn is connected to a windlass. Sufficient traction is made on the head to take up the slack and at the same time maintain the head in a slightly extended position. This should be maintained steady by an assistant. The

trunk is then bent against the primary curve. With this a certain amount of correction is obtained. It is important that

split nearly through sagittally and the space filled with cotton to form a triangular wedge. This will prevent the cut edge of the

FIG. 33.   FIG. 34.
FIGS 33 AND 34. Case II (February 15, 1935), nearly eight years after operation.

the pelvis be maintained level with the shoulders, and not rotated, during the bending process. The arm on the convex side of the curve under correction is brought to the side of the frame, while the other arm is extended over the head. At this point it is well to tilt the head about 10 degrees toward the concave side of the curve under correction, which will lessen the pull on the branchial plexus when final wedging is under way. To act as a counter-force a 6-inch muslin bandage is placed about the pelvis on the convex side of the surve to be corrected and this tied to the opposite side of the frame.

The felt pads are then held in place over each iliac crest, over the sternum, sacrum, spine, under the axillae, and about the head. The axillary pad on the convex side is

jacket from causing a pressure line on the thorax after wedging is begun.

After the placing of the felt pads, the application of the plaster is begun. One assistant starts at the pelvis while another works on the application of the head-piece. The pelvic portion should be wrapped snugly and moulded well about the crests, while the portion about the epigastrium and lower ribs should be laid on rather loosely. As the axilla is approached the bandages are drawn tighter. The head-piece must be laid on smoothly and moulded well about the jaw and occiput. After eight or nine layers have been applied the head-piece and body portions are joined together by plaster reinforcements or reverses. The hinges are then placed, the joints of the hinges at about the apex of the curve and

on a plane transverse to the trunk. Before incorporating the hinges in the plaster they should be bent to an angle of about the

into the jacket at about 3 or 4 inches below the joints of the hinges and on the concave side of the curve under correction. The

FIG. 35. Preoperative roentgenogram of Case II.

FIG. 36. Roentgenogram of Case II taken nearly eight years after operation.
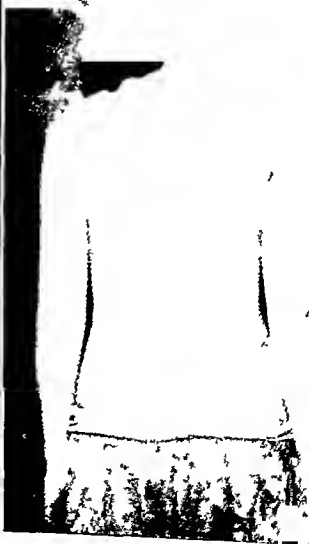
FIG. 37. Case III. A, on January 24, 1928. B, on January 5, 1935.

same degree as the primary curve so that as correction progresses they will straighten out. The turnbuckle lugs are incorporated

hardware should be well incorporated in the plaster and nearly smoothed off. Before the plaster sets some sort of a mark is

placed on the surface of the jacket to identify the joints of the hinges. (Fig. 19.)

With the patient still in this position on

maintained to dry the jacket thoroughly. A gastric upset may at times follow, but this is temporary and subsides quickly as

FIG 38. Case III, showing lateral flexibility of spine.

the frame, the thigh on the convex side of the curve is flexed about 10 degrees and abducted about 20 degrees. The remaining stockinette is cut and smoothly placed about the thigh. The lower portion of the jacket over the same iliac crest is cut upwards for about 4 inches into three or four strips about 1½ inches wide. This will relieve any pinching that may have occurred during the application of the pelvic portion. These strips are separated into several layers and folded in, one to each turn of the plaster as the spica is being applied. If greater strength is required a basswood splint may be incorporated.

The patient is then carefully removed from the frame and the edges of the jacket trimmed and bevelled. The head-piece should be trimmed wide enough to give room for the ears and the mastoid processes. (Fig. 20.)

After the patient has been returned to bed, a free circulation of air should be

the patient becomes accommodated to his new position.

The care of the jacket at this time is especially important, and nurses and interns should be instructed to inspect it frequently and keep a sharp lookout for pressure areas, which must be promptly relieved. Any area of burning or numbness complained of should be promptly investigated and if this persists a small fenestration must be made to relieve the pressure.

Wedging. After seven to ten days the jacket has become sufficiently dry and strong for wedging. A segment is removed on the convex side about 3 inches wide at the center, tapering off to the joints of the hinges. On the concave side a curved cut is made connecting the joints of the hinges and the interval between the turnbuckle lugs. The upper section is then swung on the lower and a small turnbuckle is inserted. This turnbuckle is replaced by a larger one when the limit of the smaller has

been reached, and extended as much as the patient can stand comfortably each day. The wedging is best done in the morning so

The next day the posterior portion of the jacket may be cut out to expose the operative area. The stockinette is bound to the

FIG. 39. Case III. A, roentgenogram, 1928. B, roentgenogram, 1935.

tension can be relaxed if necessary in the evening. With reasonable care a curve can be corrected and the patient kept fairly comfortable during the entire process. The maximum amount of correction obtainable can be learned from roentgenograms taken at intervals. One should direct his attention during the wedging to the arm on the concave side for evidence of pull on the cervical nerve trunks, manifested by anesthesia and weakness. If this does occur the neckpiece may be cut through and the head tilted still more to give relaxation. (Fig. 21.)

After the optimum correction and compensation have been obtained, as determined by roentgenographic examination, the upper and lower sections are well padded over the exposed portions of the thorax and plaster bandages are then used to join these into a solid unit. Plaster-covered basswood splints are then placed about the back to give further strength, securely fastened at their ends. (Fig. 22.)

edges of the fenestration with adjesive and the skin thoroughly cleansed. A silver nitrate mark is placed on the skin over a spinous process in the primary curve to serve as a guide at operation. This is accurately identified by a roentgenogram taken with a small piece of wire over the the silver nitrate marker.

Before the iodine is placed on the skin at operation, if the marker is not very clear it is well to reinforce it with gentian violet stain. The operative field is now ready for preoperative preparation. (Fig. 23.)

Selection of Area for Fusion. After the primary curve has been corrected in the wedging jacket, the selection of the proper area for operative fusion of the spine is a matter requiring the exercise of much judgment and it is here that errors are most likely to be made. At times, what should be a good result can be completely nullified by the addition or subtraction of even one vertebra at the ends of the fusion area,

especially if the patient be a growing individual.

In the ideal case, when the entire primary curve has been completely corrected, two vertebrae, one at each end, whose surfaces are parallel to each other and at right angles to a line joining their centers.

FIG. 40.   FIG. 41.

FIGS. 40 AND 41. Case IV before correction.

mary curve has been completely corrected, all the vertebrae should have their transverse axes parallel, and intersect the vertical axis of the trunk at right angles. This being the area of imbalance, it is now only necessary to maintain this position to obtain complete restoration of the alignment of the spine. After a fusion in such a case the spine outside the fusion area should balance the trunk with no external assistance.

In other cases, where complete correction has not been obtained and the transverse axis of the end vertebrae of the primary curve are not parallel to each other and at a right angle to a line joining their centers the above does not hold. In such cases to fuse only the primary curve would leave too much tilt in the vertebra at the ends of the fusion area for the compensatory curve or curves to take care of. Now if we examine the vertebrae beyond the ends of the primary curve we can, in most cases, locate

Therefore, in this case the fusion should be done to include not only the primary curve, but, in addition, sufficient vertebrae beyond the ends of the primary curve to give a level platform inferiorly, give symmetry to the trunk and enable the patient to stand erect without a list or a displacement. When the superior surface of the vertebra at the upper end of the fusion area is level the patient will hold his shoulders level with no effort.

In cases where there is severe structural wedging in the vertebral bodies of the compensatory curve or curves one must determine their flexibility before the application of the plaster jacket. This is done by means of roentgenograms taken with the pelvis elevated on the same side as the convexity of the compensatory curve, or it may be done with the spine bent laterally against the convexity of the compensatory curve. This film will show the amount of straightening the compensatory curve is

able to do when the primary curve has been corrected as much as possible. If we include in the fusion area the vertebrae at or

vertically with the first thoracic vertebra over the first sacral vertebra and the film of the spine in maximum correction also

Fig. 42.

Fig. 43.

FIGS. 42 AND 43. Case IV after correction.

beyond the ends of the primary curve whose surfaces are parallel to each other and at right angles to a line joining their centers, the compensating portions of the spine should straighten or return to the erect in order to balance the trunk. When there is structural wedging or other hindrance to the compensatory portion or portions of the spine to straighten, fusion to a level vertebra inferiorly would give overcompensation of the curve. To arrive at the correct area for fusion in these cases the following method is used:

Place the film of the patient in the jacket, when in the position of maximum correction, over the film taken with the pelvis elevated on the side of the convexity of the compensatory curve or the film taken bent against the convexity of the compensatory curve. These superimposed films should give a fair idea of how the spine will look when the treatment has been completed. The pelvic tilt film should be placed

vertical. These films are superimposed so that the lower end vertebra of the primary curve in each film coincides. The first vertebra at or beyond the end of the primary curve whose inferior surface coincides with the same surface of a corresponding vertebra in the other film should be taken as the lower limit of the fusion area. This will leave sufficient tilt in the transverse axis of the vertebra at the lower end of the fusion area to allow the compensating portion of the spine to balance the trunk. (Fig. 24.)

The Fusion Operating and After-Care. The spine is fused by the usual Hibbs method. Particular attention is paid to the apex of the curve, where the fusion is reinforced with chips from the spinous processes from the ends of the fusion area. When six or more vertebrae are to be fused the operation is carried out in two or more stages, about three weeks apart. The fenestration in the jacket is closed with

plaster after the last operation and the patient is kept in bed for twelve more weeks. After operation the patient may relax and suspend himself in the neck-piece. Careful observation will disclose points of pressure about the head and arms. Trimming to relieve this will not materially affect the correction.

When the jacket is removed roentgeno-grams are taken to determine the extent and maturity of the fusion, as well as the angle of curve. A light body jacket is then applied, with the head held lateral to the crest of the curve on the convex side in order to minimize the gravity as a deform-ing force. The patient is allowed up in this jacket. The jackets are changed at intervals of twelve weeks, the head being brought closer to the midline with each plaster change. In most cases nine months is sufficient time for the fusion to become mature enough to maintain the correction without external assistance.

CONCLUSIONS

The treatment of scoliosis by this method is long and difficult, but has given better results than the previous ones used at this clinic. This and other studies made on this subject have enabled us to state the follow-ing with reasonable accuracy;

1. If the fusion is solid and adequate in extent there will be no increase in curvature.

2. A loss in sitting height and an increase in curvature suggest a pseudarthrosis. Pain and localized tenderness also suggest a pseudarthrosis.

3. If there is no increase in curvature in the fused area but a definite increase in the curvature at the end of the fusion, with a loss of compensation, the fusion area is too short.

ILLUSTRATIVE CASES

CASE I. M. K. was admitted to the clinic August 18, 1934, at the age of 15. (Figs. 25–30.)

Wedging jacket was applied October 14, and the patient was ready for operation on Novem-ber 1. Spine fusion was done in two stages from the ninth thoracic to the fourth lumbar verte-bra. All support was removed August 8, 1935. Balance and symmetry have been restored and the spine is now geometrically straight.

CASE II. E. P. was admitted to the clinic April 18, 1927. The curve had been noticed for four months. The right thoracic curve was primary and was corrected in a wedging jacket from 27 to 23 degrees. Fusion was done from the fourth thoracic to the second lumbar vertebra and has maintained the balance and secured improvement in posture. Now the clinical appearance of the trunk is excellent.

CASE III. C. R. was admitted to the clinic at the age of 11, July 9, 1928. The lumbar curve was primary and was corrected in a wedging jacket from 37 degrees to zero. The spine was fused from the tenth thoracic to the fourth lumbar vertebra. Fusion should have included the fourth lumbar, but due to the over-correct-ness inferiorly the fusion was shortened pur-posely to give more lumbosacral flexibility and at the same time not have the spine become solid when the lower end vertebra is in an over-corrected position.

Note the excellent maintenance of correction and the good lateral flexibility of the spine below the fusion area.

CASE IV. J. R., age 15 years had a curve first noticed in 1930. She was admitted to the clinic in 1934. Correction in a wedging jacket was accomplished from 44 degrees to 11 degrees. The spine was fused in two stages from the seventh thoracic to the third lumbar vertebra. All support was removed thirty-six weeks after operation. Note the remarkable correction of the curve and the improved appearance of the trunk even though complete correction has not been obtained.

REFERENCES

FERGUSON, A. B. Study and treatment of scoliosis. *South. M. J.*, 23: 116, 1930.

HIBBS, R. A., RISSER, J. C., and FERGUSON, A. B. Scoliosis treated by the fusion operation. An end-result study of three hundred and sixty cases. *J. Bone & Joint Surg.*, 13: 91, 1931.

TUMORS OF THE MANDIBLE

SAMUEL GEORGE SCHENCK, M.D.

BROOKLYN, NEW YORK

MANDIBULAR tumors form an increasing group of neoplasms, many of which are histologically and radiographically quite unlike any found elsewhere in the body. Clinically, it is well nigh impossible to separate the various types. However, the roentgenogram is responsible for the most accurate means of recognizing and differentiating these growths. Neoplasms of the jaw are most frequently classified according to their histologic elements into epithelial, osseous and dental tumors. In this way their division is based on the structural origin of the neoplasm. From the standpoint of behavior and treatment, such a classification proves of little clinical importance.

A classification that is based on the clinical course and which suggests the proper management of the growth is certainly of more practical value. With their separation into three general groups, tumors of the mandible may be benign, malignant, or benign with the tendency to assume malignant characteristics. These clinical divisions are not hard and fast, as certain benign growths may show a tendency to recur locally when not completely eradicated. This possibility may also affect benign growth elsewhere in the body.

Mandibular tumors may accordingly be divided as follows:

I. Benign Tumors
 1. Dental root cyst or radicular cyst
 2. Follicular or dentigerous cyst
 3. Odontoma
 4. Giant cell tumor and epulis
 5. Ossifying fibroma
 6. Osteoma or exostosis
 7. Osteochondroma
 8. Hemangioma
II. Benign Tumors Which Sometimes Take on Malignant Characteristics

 1. Adamantinoma or adamantine epithelioma
 2. Radiation osteitis—from radium or x-rays—resulting in sarcoma
III. Malignant Tumors
 A. Primary:
  1. Osteogenic sarcoma
  2. Endothelial sarcoma or Ewing's sarcoma
  3. Chondrosarcoma
 B. Secondary:
  1. Carcinoma
   (a) Epidermoid carcinoma—by direct extension from adjacent structures as the mouth, skin and glands
   (b) Adenocarcinoma—by metastasis from a primary lesion elsewhere in the body.
   (c) Melanotic carcinoma
  2. Sarcoma
   (a) Osteogenic sarcoma
   (b) Melanotic sarcoma
  3. Hypernephroma

BENIGN TUMORS

These mandibular tumors are slow-growing, hard, practically painless growths which, because of the facial deformity they produce, ultimately bring the patient to the physician. They occur at any age, but more commonly in children and young adults. Treatment is surgical; complete excision with cauterization is necessary to prevent local recurrence, for which they show a tendency. They never metastasize. Irradiation has little, if any, effect.

Radicular or *Dental Root Cysts.* Dental root or radicular cysts are the most common of the dental tumors. The cyst forms in the osseous structure of the jaw about the root of a devitalized tooth following chronic inflammatory changes. Although

there are no symptoms in the early stages, the larger cysts result in a noticeable swelling and expansion of the mandible.

FIG. 1. Radicular cyst of the mandible.

The roentgenograms (Fig. 1) show a rarefied or a cystic area in the body of the mandible with clear-cut borders extending about the root of the devitalized tooth, provided the dental structure has not been removed at some time previous to the examination. There is no evidence of bone proliferation around the cystic space. These growths are to be distinguished from an ordinary apical granuloma which is usually smaller than a radicular cyst and has indistinct hazy margins, and from a follicular or dentigerous cyst. The latter is diagnosed by the presence of a nonerupted tooth with the cystic formation about its crown rather than about the apex of an erupted tooth, as occurs in radicular cysts.

Dental root cysts are occasionally multiple, involving the roots of several neighboring teeth. They then assume a multilocular appearance and must be distinguished from other multilocular cyst formations.

Follicular or Dentigerous Cysts. This type is rare and occurs usually in children under 15 years of age. The most common site is in the region of the third molar tooth. The jaw shows slow enlargement at the site of the tumor and the tooth fails to erupt. The roentgenogram reveals a unilocular

cystic area with sharply defined walls about the crown of an unerupted tooth. Bone production about the cystic space is absent.

FIG. 2. Composite follicular cysts Cysts are multilocular and one contains an unerupted tooth (arrow).

Rarely, the cyst assumes a multilocular appearance due to the superposition of several cysts involving the crowns of unerupted teeth, similar to the formation of multilocular radicular cysts. These are then spoken of as *compound* or *composite follicular cysts.* (Fig. 2.)

Pathologically, these cysts arise from the epithelium of the enamel organ during the development of the teeth.

Odontomas. These are mixed tumors occurring more frequently than follicular cysts. They are usually of bony hardness, appearing in the lower jaw of children or young adults at the site of an unerupted tooth, or from accessory tooth germs that adjoin the unerupted follicle (*composite odontoma*).

Roentgenologically, odontomas are characterized by irregular, densely classified, compact masses within a cystic space which is well delimited by normal bone structure, showing no proliferative changes. (Fig. 3.)

Giant Cell Tumor and Epulides. Central giant cell tumors of the mandible usually grow rapidly and produce early facial deformity. The Roentgen examination (Fig. 4) shows a polycystic area of bone absorption with clear-cut margins. The jaw is expanded and the cortex thinned out. At times, especially in larger lesions,

radiation therapy is necessary to augment the effects of curettage and cauterization, either to prevent spread or to control a

and their slow growth and distinct contour help to exclude a malignant condition.

Osteomas or *Exostoses.*   Osteomas are the
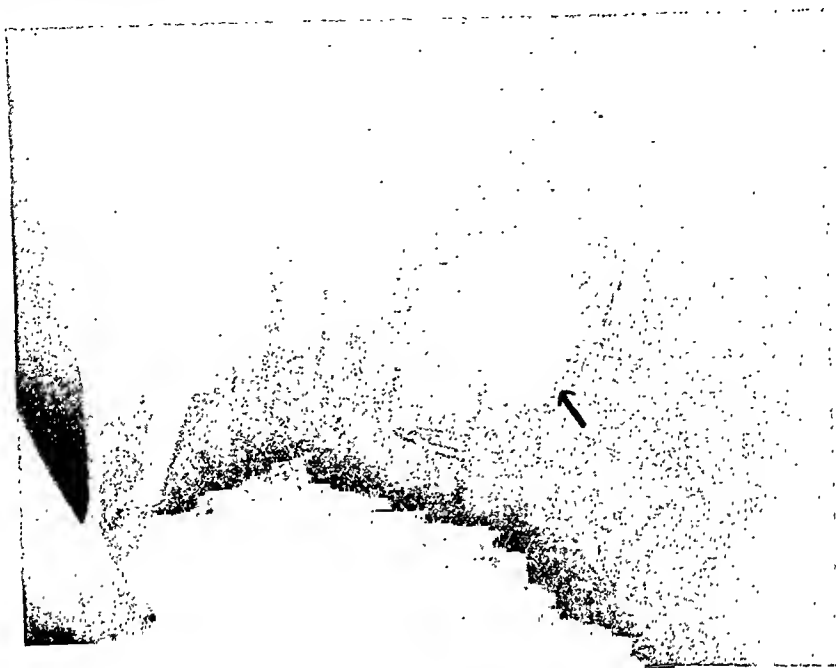
FIG. 3. Odontoma of the mandible.

recurrence. The danger of radiation osteitis must always be borne in mind when irradiating with heavy doses in and about the jaw.

There are other neoplasms which particularly affect the gums and the alveolar ridge, and are designated as epulides. They are commonly found during pregnancy when they tend to grow rapidly. The more frequent pregnancy tumors (also designated as pregnancy gingivitis or epulis gravidarum) are the angiomas and granulomas; osteomas, fibromas and alveolar giant cell epulides are less common. These gingival neoplasms exert pressure on the teeth, the displacement of which is clearly observed on the films.

Ossifying Fibromas. These benign tumors are made up of fibrous tissue, bone and sometimes cartilage. Roentgenologically, they show a coarse mottling within a regular delimiting area which appears less dense than normal bone. They have no cystic space or sharp trabeculations to confuse them with the polycystic tumors,

more differentiated and less cellular form of ossifying fibroma. The roentgenogram

FIG. 4. Central giant cell tumor.

shows a small dome-like swelling of increased density. The prognosis is good, and, in most cases, surgery is not imperative.

The more spongy growths may be chiseled from their base without the likelihood of recurrence.

FIG. 5. Hemangioma, capillary type. Note the many small cystic spaces like pouched-out areas in the bone, produced by the dilated capillaries or smaller blood spaces.

Osteochondromas. An osteoma containing a layer of cartilage is recognized as an osteochondroma. The dense mass in the roentgenogram shows a spotted or irregular appearance due to the radiotranslucency of cartilage. Treatment is the same as for osteomas or exostoses.

Hemangiomas. This type is a very rare, benign growth which may not only be present in the bone of the mandible itself, but may also be found to invade the gingival or buccal structures at the same time. As elsewhere in the body, it assumes two different forms depending on its histology. The cavernous type shows large spaces of diminished bone density, often with fine radiating spicules or long thin strands of bone in the rarefied area. In the second or the capillary form (also called the scirrhous type), the roentgenograms (Fig. 5) show many small cystic spaces which are pro-

duced by the dilated capillaries or smaller blood spaces scattered through the bone. In the more developed stage, it suggests

FIG. 6. Adamantinoma of the mandible.

the Roentgen appearance of osteitis fibrosa cystica.

Bone hemangiomas respond to irradiation, ossifying slowly following treatment.

BENIGN GROWTHS WHICH SOMETIMES TAKE ON MALIGNANT CHARACTERISTICS

Adamantinoma. Adamantine epitheliomas are unusual and interesting epithelial growths, potentially malignant, which take their origin from the enamel and dentine organs. Less common than radicular cysts, they occur more frequently than follicular cysts. Adamantinomas are slow-growing, practically painless tumors which most often affect young adults and negroes, and gradually produce facial asymmetry which ultimately brings the patient to the physician.

The roentgenogram (Fig. 6) usually shows bone destruction with the formation of a number of conglomerate cystic spaces and a thinned out cortex. A faint irregular mottling can be made out in the cystic areas. Occasionally, there is a central cystic tumor of sharp outline, not unlike that produced by the giant cell tumor or the radicular or follicular cyst. In many cases, the teeth are displaced by the expan-

sion of the growth and sometimes a tooth follicle is present in the cyst. An absolute diagnosis cannot always be made as, not

occurred from direct extension to the skull and brain.

Aberrant adamantinomas have been

FIG. 7. Osteogenic sarcoma. Note the spicule formation lying at right angles to the maxilla.

infrequently, any of the central tumors may show a similar picture. The adamantinomas produce no periosteal reaction, such as is seen in sarcoma. They have not the worn, eroded edges of the cancerous growths or the bone proliferation observed in osteomyelitis.

Adamantinomas may be cystic or solid and are frequently lobulated. Even when solid, they show a cystic or rarefied appearance on the Roentgen films. Treatment consists of curettage and cauterization. They have a strong tendency to recur when they may attain considerable size. Like the benign neoplasms, these tumors are radioresistant. Even with one or more recurrences, the prognosis is good in most cases. Metastasis is rare, but death has

recorded in the ovary (where they are teratomas), tibia and the hypophyseal duct.

Radiation Osteitis. Although this condition is not neoplastic, excessive radiation to the jaw may result in bone necrosis, which, in certain rare instances, may undergo sarcomatous changes. Such malignant development has been particularly observed in industrial workers with radium poisoning. Whether a similar complication is possible in radiation osteitis following intensive roentgen therapy remains a mooted question.

PRIMARY MALIGNANT TUMORS

Osteogenic Sarcoma. Primary involvement of the mandible with sarcoma is rare.

Almost any pathologic type has been known to occur in the maxilla. They usually grow rapidly, producing early

FIG. 8. Secondary involvement of the lower maxilla from a hypernephroma.

facial asymmetry, and are fatal regardless of treatment. Extensive skeletal metastases may occur early in the course of the disease. In the sclerosing type, growth is exceedingly rapid with the average duration of symptoms about two months. The roentgenograms (Fig. 7) show irregular dense foci of new bone production alternating with areas of bone destruction. The margin of the neoplasm shows a periosteal reaction with occasional spicule formation extending at right angles to the maxilla.

Endothelial Sarcoma. Ewing's sarcoma usually occurs in children and young adults. The symptoms of pain and swelling of the jaw are brief, frequently of one month's duration, an unusually rapid course for lesions of the jaw. The roentgenograms show irregular areas of bone destruction with or without expansion of the cortex or periosteal new bone. The findings are not characteristic. Although the lesion is radiosensitive, cures are indeed rare.

Chondrosarcoma. This tumor is an osteogenic sarcoma containing cartilage. Al-

though growth is less rapid than in the other types already mentioned with a year or more elapsing before treatment is sought by the patient, the outcome is fatal regardless of the form of therapy. Repeated local recurrence is the rule before distant metastasis takes place. Roentgenologically, an area of osteoporosis is present, extending rapidly either to the alveolar or to the lower margin of the mandible. Erosion takes place without expansion of the bone, distinguishing these growths from benign tumors.

METASTATIC TUMORS

The jaw may be invaded with malignant growth by the blood stream, lymphatic channels or by direct extension. Intra-oral epidermoid carcinoma, especially from the gums, floor of the mouth, tongue, and lips, not infrequently extends to the bone which becomes irregularly eroded by the cancerous invasion. Secondary involvement of the mandible from a primary sarcoma or carcinoma may rarely occur. The more likely primary malignant seats are the prostate, thyroid, ovary, testicle, kidney (Fig. 8), melanotic lesion usually of the skin, breast and rarely the stomach. Mandibular involvement has been observed in multiple myeloma and lymphoblastoma.

DIFFERENTIAL DIAGNOSIS

Mandibular tumors may not infrequently be confused with the following non-tumorous conditions:

1. Technical factors:
   (a) Superimposition of the hyoid bone over the mandible.
   (b) Calculus in the salivary gland.
   (c) Air in the pharynx.
2. Osteitis fibrosa cystica or hyperparathyroidism.
3. Osteitis deformans or Paget's disease (leontiasis ossium).
4. Syphilis.
5. Tuberculosis, actinomycosis, blastomycosis.
6. Radiation osteitis.
7. Osteomyelitis.

Faulty technique in regard to positioning of the patient and the tube may superimpose the hyoid bone and air-containing pharynx over the mandible, with the result that a cystic lesion of the jaw is simulated. A salivary calculus overlapping the maxilla may suggest the diagnosis of an odontoma or an exostosis. Changing the position of the patient or tube will tend to separate the shadow cast by the stone from the margins of the jaw.

Rarely, Paget's disease may be manifested in the jaw (leontiasis ossium of the face) for many years before the rest of the skeleton becomes involved. The cystic lesions observed in hyperparathyroidism may also attack the mandible, but this involvement is usually late in the course of the disease which is considerably advanced by this time and shows widespread evidence of its presence.

Syphilis in the form of a gumma may rarely involve the jaw. Tuberculosis, actinomycosis and blastomycosis have been observed to invade the mandible from intra-oral lesions which are recognized by examination and biopsy.

Radiation osteitis, the result of exposing the jaw to intensive radium and x-ray bombardment, reveals itself as a necrotic lesion. It is more apt to occur in the presence of infection, particularly of the teeth, and results in changes not unlike those observed in osteomyelitis. As a prophylactic measure, oral sepsis should be eradicated as thoroughly as possible and diseased teeth removed before executing any plan of intensive radiation therapy in the region of the jaws.

Osteomyelitis is of rather frequent occurrence and, roentgenologically, may resemble a new growth. Usually the history of a systemic reaction with an elevation of the temperature, chills and malaise helps to separate the two conditions. In infections of the mandible, a predominant symptom is pain which is seldomly very pronounced in neoplasm.

SUMMARY

A classification of mandibular tumors is suggested, based upon clinical behavior. The various types of neoplasms invading the lower jaw are described, and an attempt is made to differentiate them from one another as well as from non-neoplastic conditions.

SOME COMMON ERRORS IN DIAGNOSIS AND TREATMENT OF CHEST PROBLEMS*

J. K. DONALDSON, M.D.

Associate Professor of Surgery, University of Arkansas School of Medicine

LITTLE ROCK, ARKANSAS

PHYSICIANS and surgeons who have not followed intimately during the past few years developments in the field of chest surgery are frequently lacking in concepts of diagnostic and therapeutic possibilities in this field. This statement is not necessarily a condemnation of any part of the profession, but simply a reference to an unavoidable stage through which medicine must advance in its forward progress.

Those familiar with more modern methods of treatment and diagnosis in the chest surgery field are frequently surprised that prominent internists will often treat a patient with, for example, a basically unimproving lung abscess by so-called conservative measures for a long period, when every modern indication calls for adequate surgical bronchoscopic or surgical drainage relatively early in the disease; or that a frank lung abscess or an empyema may be treated for weeks by well known general practitioners with a diagnosis of "unresolved pneumonia."

Nevertheless, instances such as these, as every chest surgeon knows, are occurring daily. And the deplorable part of this situation is that actually very little, relatively speaking, is being done to narrow the wide breach which exists between the type of diagnosis and treatment known to men especially interested in chest pathology and that familiar to the average surgeon or physician not especially practiced in this field.

There are a number of reasons for this wide discrepancy in our efficiency, the discussion of which would not add greatly to the value of this presentation. One of these reasons, however, is that the men especially interested in following the rising star of thoracic surgery have become more or less organized in special groups. They tend to discuss their problems in special meetings and in journals devoted exclusively to the study of thoracic disorders. This course of action is a desirable one for the most part, enabling those who are pioneering the field to progress more rapidly than if chest problems were developed in less concentrated fashion. However, there is also a definite need for wider publication in journals devoted to broader phases of medicine and surgery in order that applicable fundamentals in chest problems may reach a larger portion of the profession.

A few illustrative case reports with relevant discussion are therefore presented here. There is little in the case reports or the discussion which will be of specific interest to a specialist in thoracic surgery. That there is material here, however, which should be of interest to a great many physicians and surgeons is indicated by the several errors in judgment or procedure on the part of physicians or surgeons who ordinarily are quite efficient in the handling of other human ailments. No further reason need be offered for publication of a paper which is intended, not as a contribution of original advances in the field, but as an appeal to a wider appreciation of basic factors already accepted in special chest surgery circles.

These cases represent a part of the routine work in the Chest Surgery Division of the University of Arkansas School of Medicine.

CASE I. E. G., a white male of 46 years, was admitted November 11, 1936, with a history of

"influenza" ten months previously. Following this he continued to have a moderately productive, non-odoriferous cough and some aching pains in the right chest. The chest had been x-rayed shortly after the attack of "flu" and the patient had been told the picture "showed nothing." Afterward he went to a well-known internist who told him that he had a lung abscess and *treated him with "conservative measures" for a period of several months,* i.e., until three weeks prior to his admittance to the hospital.

Upon his admission, 900 c.c. of thin, green, putrid fluid was obtained by aspiration through the ninth space in the posterior axillary line. The patient, with a diagnosis of empyema, was then transferred to the chest surgery service with the internist's recommendation that Mozingo drainage with or without rib resection be instituted. Here, 800 to 900 c.c. of pus, similar to the first, was obtained by aspiration from one area of the chest and about 400 c.c. was aspirated from another area.

The pus was smeared and cultured and *organisms belonging to streptothrix group obtained in pure culture.* After consultation with Dr. Wohlin of the bacteriology department a working diagnosis of pulmonary actinomycosis was made.

X-ray (Fig. 1) examination on November 13 showed three different fluid levels in the chest. A thoracotomy was done on November 25. Three separate and distinct cavities in the pleural cavity were opened. Thick old adhesions firmly and completely separated the right pleural cavity into two large anterior and posterior cavities by running from the dome on the right chest down the lateral wall, ending apparently by branching into an inverted Y with the two prongs forming another small but complete cavity in the lower lateral aspect of the right chest. About 700 c.c. of yellowish purulent odoriferous fluid was obtained as the septa were broken down. No sinuses were seen in the pulmonary tissue proper. Drainage tubes were installed in the pleural cavity.

Though little assistance was expected from massive doses of iodides and x-ray therapy, it was felt these latter agencies were possibly indicated as a temporary measure because of the streptothrix micro-organisms. Iodides were administered, but it was necessary to discontinue them in a few days because of intolerance. For the next thirty days, the patient with good

drainage of the chest, showed definite clinical improvement, gained weight, and felt better. On December 27 it was noted that an obstruc-

FIG. 1. Case 1. All separate pockets of pus which were present are not discernible here, but this oblique view clearly illustrates two separate fluid levels. The operative notes given in the first and fourth cases reported clearly signify the necessity of realizing that several separate pus pockets are sometimes present in empyema; and that all these pockets may not be drained by the simple insertion of a single tube into the pleural cavity.

tive type of inspiration, moderately labored had been noticeable for three or four days and coughing had increased. Definite sonorous and sibilant râles were heard over the right chest. The cough was essentially non-productive and the general condition was fair.

Drainage from the chest was decreasing, but apparently the lung was not expanding. On December 30 the cough became more severe. Morphia was necessary for its control at times. The breathing was labored and of a type which indicated partial obstruction of the left main bronchus. It was believed that granulation tissue was filling in from the right bronchus or that possibly a tumor was present. The patient was obviously losing ground and other procedures were indicated, the first of them a bronchoscopic examination. It was in our minds at this time to confirm or rule out new growths or remove any granulation tissue which might be associated with the suspected actinomycosis of the lung, and then consider pneumonectomy.

Bronchoscopic examination done on January 5, 1937 by Dr. Paul Mahoney showed the "carina markedly thickened, the right main

of fungus infection of lung. (Figs. 2 and 3.) A guarded prognosis was offered. On September 13, the general condition appeared essentially
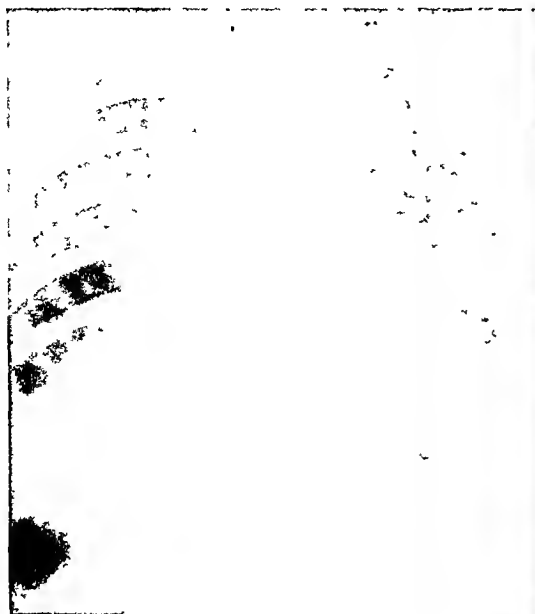
FIG. 2.

FIG. 3.

FIGS. 2 AND 3. Case II. The somewhat irregular shadows partially obscured by the heart shadow indicate the area of atelectasis and pneumonitis. These complications followed the blocking of the bronchus by inflammatory and granulation tissue which formed about the aspirated foreign body. Bronchoscopic examination should have been done earlier in this case. Purely symptomatic treatment was continued too long under the diagnosis of lobar or "unresolved" pneumonia.

bronchus filled with granulation tissue and necrotic. The left was partially filled with tissue, but showed no evidence of necrosis." Tissue removed from the left main bronchus on biopsy revealed grade 2 carcinoma. The disease was so extensive that a pneumonectomy was contraindicated.

The patient died on April 3, and autopsy showed carcinoma of right lung, originating probably in the right main bronchus.

CASE II. C. G., a white female, aged 43 years, was admitted September 10, 1937, complaining of general weakness, cough, moderately profuse and odoriferous expectoration of thick, yellow, purulent sputum, high fever, and pain in the left chest. She had been in good health until about four weeks previously, at which time she had an upper respiratory syndrome which she believed was caused by pneumonia.

Her temperature on admittance at 12 noon was 104 degrees and at 12 midnight 100.5. The following day a tentative diagnosis of lung abscess of the left lower lobe or tubercular pneumonia was made, with a third possibility

the same, but the diagnosis of lobar pneumonia in the left lower lobe and beginning pneumonia in the right was given. Active tuberculosis possibly underlay the pneumonia. A week later the temperature was running a septic course, and the patient's condition seemed to be slowly becoming worse. The urine on September 22 showed acid-fast organisms and guinea-pig inoculation was ordered.

The chest surgery division was asked to see this patient on the following day. As far as we could determine from the patient and the intern, the amount of sputum in twenty-four hours was from 200 to 300 c.c., having perhaps diminished slightly in amount during the past week. The sputum was slightly greenish and thick, but not particularly odoriferous. No separation into layers occurred upon standing. The history of good health up to about eight weeks before; the onset of a "cold," with no aching of the bones but some semiproductive cough, loss of pep and malaise; the gradual approach of the acute stage, with high septic temperature, productive purulent cough, some pain in the left chest low down; the respira-

tion, general appearance, and the laboratory findings indicated several possibilities probably not of pneumococcic etiology. Tuberculosis might possibly have been present, but we doubted its importance as an immediate therapeutic or etiologic concern. The markedly increased breath and voice sounds over the greater part of the left lung base region posteriorly, the diminution in resonance but lack of marked dulness, and the history suggested pyogenic involvement of the left lower lobe in the region of the abnormal physical and roentgenologic findings, with possibilities of other underlying pathology associated with the pyogenic involvement. There was no history of aspiration of a foreign body, nor any factor suggestive of embolism. The history would cast a doubt upon any pulmonary plugging from bronchial secretions playing a prominent initial part in an atelectasis. An atelectatic phenomenon, however, associated with plugging secondary to possible aspiration, bacterial and mucous collection, or associated with new growth could not be ruled out.

The immediate consideration of practical importance appeared to be the lower lobe involvement of pyogenic nature which had assumed the importance of a lung abscess. The indicated diagnostic procedure was clear: a bronchoscopic examination was definitely necessary, to be followed, if indicated, by the injection of iodized oil. Good postural drainage would possibly be increased by this procedure. At the same time further useful diagnostic findings might present themselves through the bronchoscopic examination. Should a new growth or other visible complicating pathology appear to complicate the pyogenic picture the therapeutic considerations would, of course, be partly cleared. In the event that the case was one of uncomplicated abscess, conservative treatment, with or without repeated bronchoscopic drainage, could be pursued until and if surgery became indicated.

Following this consultation no immediate change in treatment was made, the attendants having decided on further delay with additional observation. From September 28 to October 7 little change occurred, but on the latter date a change of service occurred. The new attendant diagnosed "unresolved lobar pneumonia" of the left lower lobe.

One week later he ordered a bronchoscopic examination. *A small piece of wooden stick about ¼ inch long was removed from the suppurating*

granulation tissue in the left main bronchus. It was then found that the patient had a habit of chewing sticks and matches. Following procurement of drainage by removal of this foreign body and the granulation tissue about it, the patient entered immediately into an uneventful recovery.

Case iii. L. D., a white male, 17 years old, was admitted April 16, 1937 with a history of "pneumonia." This had begun about 6 weeks previously, but he had never recovered. There had been a loss of 25 pounds in weight. All signs and symptoms pointed to massive empyema (Fig. 4.)

Recommendation had been made that drainage tubes be inserted through small thoracotomy openings. The day after admission the chest was aspirated in a right interspace of the mid-axillary line by the resident surgeon. No pus was obtained. Aspiration was then done at the angle of the scapula and a few c.c. of thick, creamy, greenish pus was obtained. Another tap the next day was unsuccessful, and the possibility of encapsulated pockets was mentioned. On April 19, ½ to 1 ounce of purulent fluid was aspirated in different areas, indicating the presence of pockets. Exploratory thoracotomy was indicated.

On April 20 about 5 cm. of each of the eighth and ninth ribs was resected in the posterior axillary line. The parietal pleura was opened and several different, firmly encapsulated pockets of pus were found in the right pleural cavity. There was one large interlobar encapsulation of pus between the middle and lower lobe which contained 8 ounces or more of clotted purulent odoriferous exudate resembling very much in appearance dirty clabber milk. Some of the pockets were filled with dirty grayish pus, others with pus somewhat thicker and whiter. All septa of the separate pockets were broken down and closed drainage was instituted. The patient had a satisfactory recovery without further chest complications and the lung expanded to its full capacity before his discharge on May 20, 1937.

Case iv. H. B., a white male of 62, was admitted January 1, 1938 with a complaint of a dry cough which had begun about one year previously.

Eight months prior to admittance he quit work and was sent to a sanatorium with a diagnosis of tuberculosis without positive sputum. He was then sent to the City Hospital for a bronchoscopic examination, which re-

vealed a rather firm mass in the right main bronchus, approximately 1 inch distal to the primary bifurcation. The bronchus lumen was

FIG. 4. Case III. Too frequently the clinician's diagnostic curiosity is satisfied with a roentgenologic report of empyema. Though a massive empyema may be present, as is true here, it is always necessary to inquire carefully into the cause. In this case the empyema followed pneumonia. In Case 1 we obtained a similar picture and an x-ray report of empyema, but the primary etiologic factor was a bronchial carcinoma.

almost entirely occluded. A biopsy revealed squamous cell carcinoma. (Fig. 5.)

After preliminary pneumothorax, pneumonectomy was attempted on January 14. The right mediastinal pleura was reflected after the method of Rienhoff, and an excellent exposure of the right hilus was obtained. The large primary trunks of the pulmonary veins were rapidly made visible without any untoward effects. At this stage it appeared that individual ligation of the structures of the hilus would be relatively easy. The deep structures of the hilus, however, were thoroughly caked with inflammatory and neoplastic tissue and were so tenaciously buried within this tissue that the arteries and veins could not be isolated without hemorrhage. We were determined, however, to do a clean glandular dissection of the hilus with removal of the lung and hilus at the bifurcation of the trachea. This was the patient's only chance for recovery. This method appeared pref-

erable to mass ligation with removal of the lung, which meant leaving carcinoma throughout an undisturbed hilus. Though the lung was

FIG. 5. Case IV. A roentgenologic shadow which could be produced by various causes. If diagnostic curiosity had been satisfied with a diagnosis of lung abscess without inquiry into the specific cause, the bronchogenic carcinoma which was visualized through the bronchoscope and which blocked the lumen of the bronchus giving atelectasis and lung abscess, would probably never have been discovered.

finally removed, a considerable amount of hemorrhage was encountered. The patient was given a blood transfusion on the table, but he died shortly after the lung was extirpated.

The bronchogenic carcinoma was complicated also by a pulmonary abscess.

DISCUSSION

In the first case mentioned several errors in treatment were made, and the disregard of proper diagnostic procedures was noticeable. Internists must appreciate that thoracic surgeons have amply proved that a patient with a lung abscess should usually be treated by surgical means if satisfactory improvement in the abscess proper does not occur relatively soon under such conservative measures as postural drainage, attempts at specific drug therapy or bronchoscopic maneuvers. Most of all, one

must realize that a lung abscess may be produced by a variety of factors; unless it is clear that it is of primary pyogenic origin, *one must always look for the mechanism of origin.* Aspirated foreign bodies, carcinomata originating in the large bronchi and partially or completely obstructing and producing atelectasis, stasis and abscess, are relatively frequent causes.

Another interesting factor in the first case was the fact that organisms belonging to the streptothrix group were obtained in pure culture. This led us to believe that an actinomycotic infection was present. This pure culture was secondary, apparently, to a bronchogenic carcinoma. It may be that different organisms were present in the other pockets of this pleural cavity. The accompanying bacteriologic findings illustrate how easily one can become confused. We could have overlooked the entire trend of the pulmonary events, i.e., bronchogenic carcinoma causing lung abscess and, finally, empyema with multiple encapsulations.

I saw recently in consultation a similar case in which spinach swallowed by the patient came out through the incision remaining after an operation for a lung abscess. It had not been previously appreciated that carcinoma of the esophagus was the original lesion. The lung abscess had occurred secondarily, as a result of bacteria and erosion traveling outside the large bronchi into the lung.

Early Bronchoscopic Examination. In the second case cited above there was a history of onset of the illness *twelve weeks* prior to bronchoscopic examination, with a temperature rising to 104 degrees, medical attendants were prone to refer to the pathology as being lobar or "unresolved" pneumonia. Any pulmonary infection caused by the common pyogenic organisms, regardless of whether ordinary lobar or bronchial pneumonia were at the onset an etiologic agent, which has persisted four weeks or longer, must be looked upon as having perhaps assumed the status of more complicated pathology. An incipient lung abscess may be present, or a

complicating empyema. Recognized diagnostic and therapeutic measures should be instituted promptly. Ordinary symptomatic medication and watchful waiting are useless. *"Unresolved pneumonia" when used in such manner as above becomes a vague and loose term of doubtful scientific value.*

In Case II, the simple procedure of bronchoscopy was easily completed and a small piece of stick imbedded in purulent obstructing granulation was removed from the left bronchus. Though the patient then made a rapid and uneventful recovery, proper diagnostic and therapeutic effort had been delayed four weeks longer than necessary. This could easily have meant the difference between a full blown abscess and a simple pneumonitis. Furthermore, even now it is impossible to say that the delay has not predisposed toward a future bronchiectasis of troublesome nature because of damage which may have been done to the involved bronchi.

Multiple Encapsulations in Empyema. The third case cited (and the first in certain aspects) is illustrative of a type we frequently see improperly treated. A most inexcusable error, occasionally made, is to allow an empyema complicating a pneumonia to remain too long undiagnosed. In Case III the diagnosis was made by the attending physician before the patient was admitted to the hospital and four weeks after the onset of the pneumonia. This is a much earlier diagnosis than some.

The point to be stressed here is that *multiple encapsulations of pus were present in these two chests.* Had the recommendation of simple drainage been followed, *only one pocket* would have been drained and the patient's convalescence would have been delayed until other pockets had been suspected and properly treated. The patient would have been subjected to additional operative procedures because the full degree of pathology had not been ascertained or suspected before surgery was instituted. Though Lillienthal, in 1922, called attention to the necessity of doing exploratory thoracotomies in many cases

of empyema, particularly the neglected ones, many cases of multiple encapsulations of empyema undergo simple open

FIG. 6. A patient in whom it was necessary eventually to remove ribs over a chronic empyema cavity to allow collapse of the soft parts. A cure was obtained.

drainage, without effort being made to ascertain if multiple pockets are present. Convalescence may be prolonged for many weeks, and death may even occur, before the attendant realizes that pockets are present which have never been drained. Although one prominent thoracic surgeon disagrees somewhat with this attitude, our experiences uphold Lillienthal's point of view. This does not mean, of course, that exploratory thoracotomy is indicated in every case of empyema, but that it is imperative to keep in mind the possibility of multiple encapsulated pockets.

An exploratory thoracotomy may be done with resection of only a bit more rib than is necessary for simple tube instillation. It is frequently necessary to insert only two fingers through the thoracotomy opening to break down different pockets. However, unless this is done sufficiently early when indicated, thoracoplasty may finally become necessary for the obliteration of an old empyema cavity. The neglected adhesions become so thick and profuse that decortication of the lung and ultimate expansion become impossible, even though the adhesions are freed again and again. This end result in another of our patients is illustrated by an x-ray plate showing final recovery after removal of overlying ribs following several less radical attempts at cure.

Necessity of Differentiating between Cough and Other Symptoms Caused by Tuberculosis or Bronchogenic Carcinoma. Since bronchogenic carcinoma is no longer an utterly hopeless disease, it is no longer permissible to delay or avoid indicated procedures. Lipiodol injections and bronchoscopic examinations are necessary in patients presenting unexplained cough, possible blood-tinged sputum, and other signs and symptoms so frequently attributed to tuberculosis, unless one has well-founded scientific reasons for the diagnosis of uncomplicated tuberculosis. A typical x-ray picture, typical crepitant râles, and, if possible, a positive sputum should be obtained before the attendant dismisses too long from his diagnostic horizon, carcinoma or other non-tuberculous pulmonary afflictions. About 80 to 90 per cent of primary lung carcinomas seem to originate in the large bronchi and a great many of these may be visualized by the bronchoscope early in their development. Biopsy may very frequently be done without excessive danger or discomfort to the patient. If such neoplasms are diagnosed in the early stage it is possible that expert thoracic surgeons may cure, even at this time, 50 per cent(?) or more of lung (bronchogenic) carcinomas by pneumonectomy. X-ray treatments are apparently of no value at present and radium implantations are less desirable than surgery in most cases.

SUMMARY

The above discussion of chest problems and the illustrative case reports are an index to the frequency with which physicians not especially interested in this field

are contributing to mortality and morbidity by their failure to pursue proper diagnostic and therapeutic procedures. These errors are made not only by men who neglect altogether to read medical journals, but frequently by otherwise generally efficient physicians and surgeons as well.

Some of the most common errors in diagnostic and therapeutic procedures which are made by the general profession are as follows:

1. Failure to request bronchoscopic examinations in doubtful cases such as those discussed above. Lack of appreciation of the pathology, physiology, and varying etiology of atelectasis and lung abscess seems in particular to be noteworthy, the term "unresolved pneumonia" being used altogether too frequently.

2. Failure to consider the various potential etiologic factors in empyema, thereby allowing diagnostic curiosity to be too frequently satisfied with an x-ray report of empyema is common. It is necessary to realize that lung abscesses from various causes, including lung carcinomata and aspirated foreign bodies, may have been present previous to the empyema.

3. Failure to determine whether or not multiple encapsulations are present in neglected cases of empyema and to treat these pockets by open thoracotomy with breaking of the septa instead of simple insertion of a single drainage tube with consequent drainage of only one pocket.

4. The still too frequent tendency to classify all chronic coughs and accompanying symptoms as tubercular; and allowing individuals to go for weeks or months "awaiting developments" when early pursuit of procedures, such as lipiodol injections and bronchoscopy, might be life-saving, particularly in early bronchogenic carcinoma.

5. Failure to appreciate that thoracic surgeons now have ample statistics proving that pneumonectomy, lobectomy, pneumonotomy and thoracoplastic procedures judiciously used are tremendously valuable in otherwise hopeless chest conditions including, in addition to those disorders mentioned above, far advanced bilateral cavernous pulmonary tuberculosis.

BILATERAL EMPYEMA*

FURMAN ANGEL, M.D.   AND   HAROLD A. MENGLE, M.D.

Surgeon-in-Chief, Angel Hospital   Assistant in Surgery, Angel Hospital

FRANKLIN, NORTH CAROLINA

ARTICLES dealing with the subject of empyema have become gradually limited to a discussion of the methods of treatment, drainage, etc., the pathology, pathogenesis and etiology having been well established. In the hands of competent men, accustomed to treating the disease, the resultant mortality is low and the whole subject is well standardized. One variety of empyema of the pleural cavities, however, remains sufficiently rare to warrant further consideration. This is bilateral empyema, which may be considered unusual when one reflects that many surgeons have never seen it at all, or else find their experience limited to one or two cases in civil practice. This is borne out by a rather cursory examination of the literature, in which practically all the reports are limited to one or two cases each, with the exception of the large series reported by Dunham[1] in 1917, from the U. S. Army Camps. Table I gives a list of authors and number of cases reported in the literature since 1905.

The first mention of bilateral empyema was apparently made by Charrier[2] in 1855, who saw many cases following an epidemic of puerperal sepsis. Isolated cases have been reported from time to time since then, and these have been summarized and studied by Hellin in 1905,[3] Fabrikant[4] in 1911 and by Keyes[5] in 1931. Our own experience recently in dealing with such a case has prompted this report.

Keyes in his excellent study of this subject reported three cases from the New York Hospital and collected thirty-eight cases from the literature of the previous ten years. He defined bilateral empyema as "a condition in which pus is found in both pleural cavities at or about the same time." There is usually a common etiologic background, since in most cases the contralateral infection may be regarded as a complication of the original unilateral empyema. The cases reported all seem to be acute, including our own.

Incidence. Estimates as to the frequency of bilateral empyema among all cases of empyema vary from 0.6 per cent to 42 per cent. Keyes[5] reported three instances in 495 cases—0.6 per cent; Geitz[6] reported five out of 588 cases; Lord[7] in Osler's "Modern Medicine" found five in 248 cases—2 per cent; Hellin[3] gives the incidence as 7.7 per cent, collecting 113 out of 1,448 cases. Graham[8] in his "Surgical Diagnosis" states that its incidence "is probably less than 5 per cent"; Da Costa,[9] in his textbook, says "double empyema is an extremely fatal condition. In civil practice I have seldom seen it." Following influenza, especially of the epidemic type, the incidence is very much higher. Stone[10] reported nineteen cases out of 100 in 1918—19 per cent; Dunham[1] found 253 out of 603 empyemas to be bilateral in the U. S. Army camps in 1917—42 per cent.

In children, the incidence of bilateral empyema may be somewhat lower, if one includes the cases in adults following influenza. Holt and Howland,[11] in their textbook, state that in children empyema may be bilateral in about 3 per cent, "oftener in infants." Goldberg and Wexler,[12] who in 1936 reported one case in a child and reviewed the literature to 1910, concluded that the incidence (from the collected cases) was 2.28 per cent. In 1933 Mueller and Mogavero[48] reviewed the literature and reported a total of 312 cases collected up to the time of their article. Since then there have been several sporadic

cases reported,[12,49,50,51,52] including an interesting case by Stenbuck[51] in which, in addition to a bilateral empyema, there was present a purulent pericarditis which was treated by open drainage.

Sex. Among the cases reported, males predominate over females.

Etiology. Pneumonia is the chief etiologic agent, being responsible for over three-quarters of the cases. Seventy-five per cent of these pneumonias were bronchopneumonias. The bronchopneumonias following the exanthemata gave rise to an especially virulent form with very high mortality. Fabrikant[4] reported 20 per cent of his cases of bilateral empyema to be "primary," although this is rather difficult of conception. More rarely, pyemia, tuberculosis, actinomycosis, surgical operations and puerperal sepsis have been recorded as primary causes. Curtis and Bowman[46] in 1932 reported a case in which bilateral staphylococcic empyema resulted from a carbuncle.

Bacteriology. In approximately 70 per cent of cases the pneumococcus is the causative organism. The hemolytic streptococcus is the usual offender in the remainder of the cases, except for scattering reports of other organisms, such as staphylococci, actinomycosis, B. tuberculosis, etc. More rarely, different organisms have been reported in the two sides (Keyes[5]). In the bilateral empyemas following influenza, the usual percentage of pneumococcus 75 per cent—hemolytic streptococcus 25 per cent was reversed, streptococci being found in 73 per cent and pneumococci in 23 per cent of the cases.

Pathology and Pathogenesis. The pathology is in no way different from that of unilateral empyema. In about 50 per cent of the cases, the double involvement was preceded by a bilateral pneumonia. It is especially prone to occur in the weak, debilitated individual, whose resistance has been markedly diminished by the initial empyema. In those cases in which bilateral empyema followed unilateral pneumonia, most of the writers postulate either meta-static infection or a pneumonic focus so small as to have been overlooked. That this does not occur more often undoubtedly is due to rapid formation of antibodies and

TABLE I

CASES REPORTED IN THE LITERATURE SINCE 1905

Author	Year	No. Cases Reported
Hellin[3]	1905	113
	1907	
Fabrikant[4]	1911	118
Edmund[13]	1910	1
Corner and Grant[14]	1911	1
Gand and Poissonnier[15]	1911	1
Bozzetti[16]	1913	1
Zingher[17]	1913	1
MacKenzie[18]	1914	4
Bunts[19] .	1914	1
Lund and Morrison[20]	1916	1
Cafritz[21]	1918	1
Norrlin[22]	1919	1
Gundrum[23]	1920	1
Durham[24]	1920	1
Glenn[25]	1920	2
Jchn[26]	1921	2
Ladd and Cutler[27]	1921	3
Stenrus[28]	1920	1
Beck[29]	1921	1
Gomez[30]	1921	1
Schweizer[31]	1921	1
Andrenelli[32]	1923	1
Ormos[33]	1924	1
Auer[34]	1925	1
Hedblom[35]	1925	1
Mackey[36]	1925	1
Matthews[37]	1927	1
Graves[38]	1928	1
Scanlan[39]	1928	1
Dodds[40]	1928	2
Tixier and De Seze[41]	1928	1
Ravnitsky and Bogin[42]	1930	5
Manson[43]	1930	1
Keyes[5]	1931	3
Macera[44]	1931	2
Guerin[45]	1932	1
Curtis and Bowman[46]	1932	1
Steinke[47]	1933	8
Mueller and Mogavero[48]	1933	1
D'Arcy McCrea[49]	1934	1
Goldberg and Wexler[12]	1936	1
Harrington, Dorsey, and Strohl[50]	1935	1
Stenbuck[51].	1936	1
Willis[52].	1938	1
Snow[53]	1935	1
Post influenzal cases		
Stone[10]	1918	19
Dunham[1] .	1917	253
Total cases reported.	. .	455

FIG. 1. Graphic chart showing record of pulse and temperature fluctuation during the patient's illness.

local tissue immunity in the majority of cases. In all these cases of double involvement, there must have occurred some

FIG. 2. Radiograph taken during the first week of illness, showing the bilateral pneumonic involvement, further advanced on the left.

interference with the immunity process, a condition which may follow either increased virulence of the invading organism or decreased resistance of the host, or both. Increased virulence of the causative organism was undoubtedly responsible not only for the high incidence of bilateral empyema in the influenza pandemic but for the high mortality as well.

While the time of appearance of the two empyemas may be simultaneous, in the majority of cases one side precedes the other by a few days to several weeks.

Prognosis. The mortality as reported by the various authors varies from 19 to 44 per cent. The lower mortality rates are obtained when dealing with the pneumococcic type, while the mortality from the streptococcic type is exceedingly high.

Treatment. In general the treatment is the same as for unilateral empyema. If the bilateral involvement comes on simultaneously, it is probably better to confine one's efforts to aspiration of the pus as long as possible until the mediastinum has
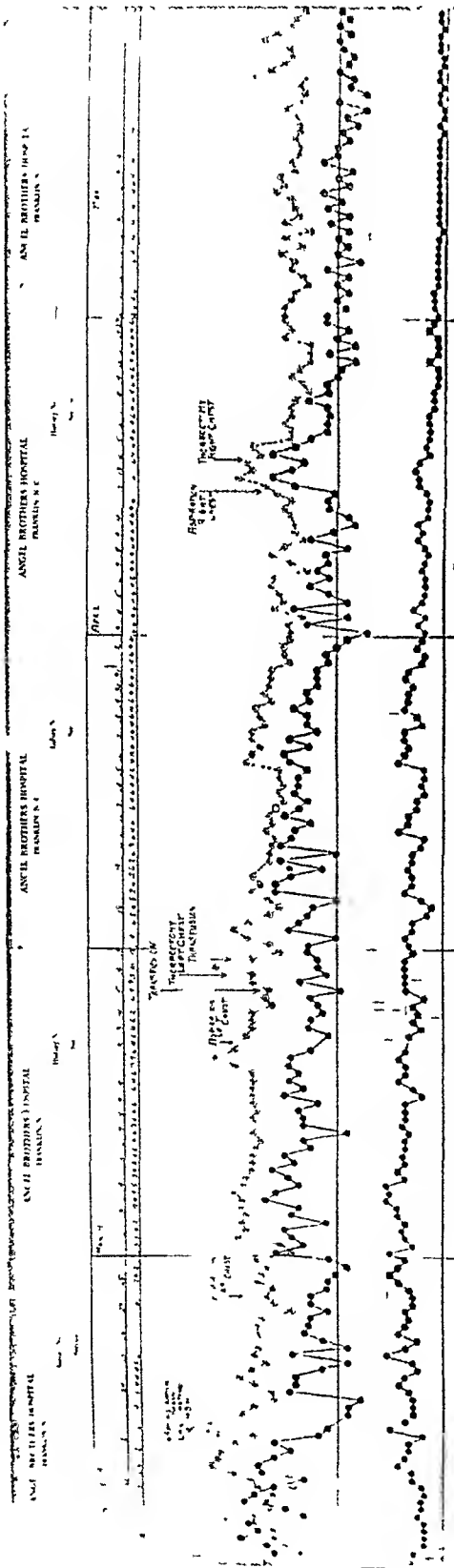
had time to become relatively fixed by adhesions or induration. In the streptococcic infections, this is notoriously slow in

CASE REPORT

D. S., male, age 38, a forester, was admitted to Angel Hospital February 20, 1938. Chief

FIG. 3. Radiograph taken April 14, 1938, revealing well-developed empyema on right side, and drainage tube in situ on left. (Unfortunately, the film showing the left-sided empyema before drainage was damaged too badly to be reproduced.)
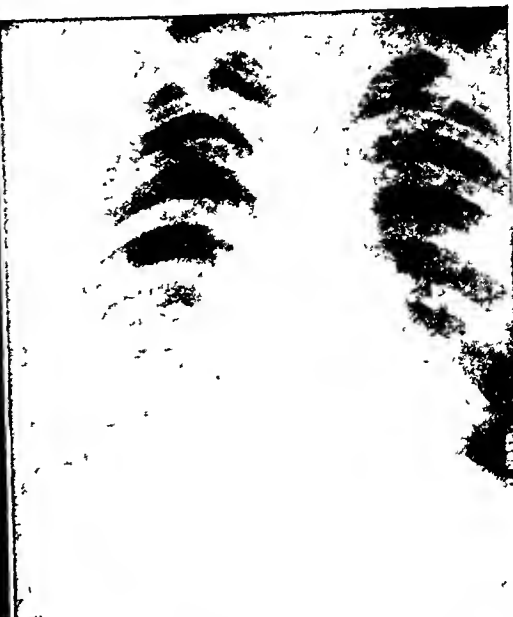
FIG. 4. Radiograph made May 15, 1938, showing both sides of the chest clearing rapidly.

development, usually requiring two to three weeks. Fortunately, in these cases the pus remains thin and is more easily aspirated. When thick pus is present, so that aspiration is difficult or impossible, little is gained by delay and thoracotomy with closed drainage, maintaining a negative intrapleural pressure, should be instituted.

Keyes[5] has ably discussed the question of simultaneous bilateral thoracotomy and concluded that the fears expressed by the earlier writers as to the attendant hazards were unwarranted. He found that "a total of 53 simultaneous or nearly simultaneous thoracotomies had been performed without a single operative death, and with eventual recovery of all but one patient . . . all the above . . . were performed under the 'open' method, . . . less favorable . . . than the safer closed method. . . . Delay in opening the second side, therefore, seems to accomplish little, but to weaken the powers of resistance."

complaints were stabbing pain in the left chest, exaggerated on breathing, fever and weakness. Three days previously he had developed a cold and cough to which he had paid scant attention. Twenty-fours before admission he had done some rather strenuous labor, becoming overheated and subsequently chilled. Shortly thereafter he had developed fever, malaise, and sharp stabbing pains in the left chest, exaggerated by respirations. He continued to have a slight unproductive cough.

Physical examination revealed a strongly developed adult male, with some dyspnea and slight cyanosis, requiring elevation of the head of the bed for comfort. Temperature was 102°F., pulse 100, respirations 36. There was playing of the ala nasi. Examination of the chest revealed a slight pleural rub, heard over the seventh rib, midaxillary line, and slight impairment to percussion over the left lower chest posteriorly. The heart sounds were normal, the rate rapid but regular. The next morning, there was dulness to percussion over the left base posteriorly and in the midaxillary line, with harsh bronchial breathing. There were numerous coarse râles scattered throughout the chest. The sputum continued scant and grayish in color. It was stained and examined

for microorganisms, but nothing conclusive was found.

An ophthalmic test for serum sensitivity was carried out, and 20,000 units of anti-pneumo-

Ephedrine was administered in an effort to eliminate any asthmatic factor and to stimulate the circulatory system. Digitalis was administered in order to bolster up the heart.

FIG. 5. Patient shortly before his discharge from the hospital (photographs taken May 15, 1938).

coccic serum, Types I and II, diluted with 150 c.c. of normal saline solution, were given intravenously, on an empiric basis. Despite the negative ophthalmic test, a violent reaction to the serum ensued, with high fever, very rapid pulse, chill and cyanosis. This required repeated injections of adrenalin and oxygen inhalations by nasal catheter.

Further inquiry into the patient's history revealed that he was a mild asthmatic, and "wheezed whenever he caught cold." Subsequently coarse moist râles continued scattered throughout the chest, dulness to percussion persisted at the left base posteriorly and he continued to have a high fever and rapid pulse.

On February 27, it was noted that the cyanosis was increasing despite continuous oxygen therapy, the sputum was increasing in amount and there was now absence of breath sounds over the left lung posteriorly, with flatness on percussion, mucous râles continuing over the remainder of the chest. The left chest was tapped in the seventh interspace, posterior axillary line, and 150 c.c. of cloudy yellowish fluid withdrawn. Microscopic examination of this fluid revealed numerous leucocytes but no microorganisms. The pulse rate continued to rise and became somewhat irregular.

On March 8, x-ray examination revealed either a beginning lung abscess or an interlobar empyema on the left side. The right lung showed some suspicious mottling but no definite areas of consolidation. At this time the patient's general condition was very poor. He was toxic, disoriented and cyanotic. Inductotherm treatments were given over the left chest, in an effort to encourage localization and resolution of the possible lung abscess. Four days later aspiration of the left chest was carried out in the seventh interspace, posterior axillary line, and cloudy yellowish fluid was obtained. On March 15, pus was obtained by aspiration for the first time very deeply situated, and seemingly directly behind the heart. Because of his desperate condition he was given 350 c.c. of whole blood by direct transfusion in the afternoon and thoracotomy was performed in the patient's room, on the following morning; 1,500 c.c. of thick, yellowish-green pus, without odor, were obtained. Another transfusion of 320 c.c. of whole blood was given the same day.

Irrigation of the cavity with normal saline and Dakin's solution was begun on March 20, and the patient began to improve. However, he continued to run an irregular fever and his pulse rate remained elevated. Coarse mucous

râles persisted over the right chest, he had a productive cough and remained very weak and toxic.

On April 12 flatness on percussion and absent breath sounds were noted over the right base posteriorly. Aspiration revealed cloudy yellowish fluid. X-ray two days later revealed empyema of the lower right chest, with displacement of the heart of the left. The left chest was still cloudy, and the drainage tube on the left side was still functioning, copious drainate still going on. Thoracotomy (open) was carried out on the right side the same day and approximately 1,500 c.c. of thick, yellowish-green odorless pus was obtained. From then on, the patient's convalescence was uneventful, albeit slow, and he was finally discharged from the hospital May 24, 1938. At that time the left chest was healed, and the sinus in the right chest was still draining slightly.

DISCUSSION

This case is fairly typical of those already reported in the literature. It is our belief that the severe serum reaction which this patient sustained produced an overwhelming of the immunity process, which led to the ultimate bilateral involvement. This is further borne out by the fact, not mentioned above, that the total leucocyte count on admission was 18,000, while the day following the serum reaction it dropped to 8,000. Also, the patient's asthmatic background undoubtedly interfered with the development of the local tissue immunity necessary to accomplish a successful outcome of the pneumonic process. It may also be worthwhile to point out that both pleurae were draining simultaneously, with open drainage, without any ill effect to the patient, a fact already emphasized by Keyes.[5]

SUMMARY

The incidence, mortality and pathogenesis of bilateral empyema of the pleura are briefly discussed.

A partial review of the literature is appended.

One additional case is reported.

REFERENCES

1. DUNHAM, E. K. Empyema, p. 163. Government Printing Office, Washington, D. C., 1924.
2. CHARRIER. Thèse de Paris, 1855. Quoted by Hellin.[3]
3. HELLIN, D. Bilateral empyema. Berlin. klin. Wcbnscbr., 42: 1415, 1905; Bilateral empyema. Arch. f. klin. Chir., 82: 866, 1907.
4. FABRIKANT, M. B. Bilateral purulent pleurisy. Deutsche Ztscbr. f. Chir., 108: 584, 1911.
5. KEYES, E. L. Bilateral empyema. Ann. Surg., 93: 1050, 1931.
6. GEITZ. Surgical treatment of pulmonary conditions. Tenth Congress of the Society of the North, Copenhagen, 1913. Quoted by Norrlin, L.[22]
7. LORD, F. T. In Osler's Modern Medicine, 4: 260, 1927. Lea & Febiger, Phila.
8. GRAHAM, E. A. Surgical Diagnosis, 1: 126, 1930. Saunders, Phila.
9. DA COSTA, J. C. Modern Surgery. 9th edition, p. 878. 1925. Saunders.
10. STONE, W. J. Management of postpneumonic empyema. Am. J. M. Sc., 158: 1, 1919.
11. HOLT, L. E., and HOWLAND, J. Diseases of Infancy and Childhood. 8th edition, p. 444. New York, 1923. Appleton.
12. GOLDBERG, S., and WEXLER, H. Bilateral empyema in children. Arch. Pediat., 53: 80 (Feb.) 1936.
13. EDMUND, W. Double empyema in a child. Brit. M. J., 1: 1052, 1911.
14. CORNER and GRANT. 1911. Quoted by Keyes.[5]
15. GAND and POISSONNIER. Case of double empyema in a child. Rev. gén. de chir. et de thérap., 25: 184, 1911.
16. BOZZETTI, L. Gazz. d. Osp., 34: 129, 1913.
17. ZINGHER, A. Case of bilateral empyema thoracis. Am. J. Obst., 68: 599, 1913.
18. MacKENZIE, K. A. J. Double and anomalous forms of empyema. Boston M. & S. J., 171: 424, 1914.
19. BUNTS. 1914. Quoted by Keyes.[5]
20. LUND, F. B., and MORRISON, H. Boston M. & S. J., 175: 606, 1916.
21. CAFRITZ, E. A. Case of bilateral empyema treated successfully by operation. Bull. Univ. Maryland School Med., 3: 216, 1918–1919.
22. NORRLIN, L. Double metapneumonic empyema. Acta chir. scandinav. 1 (Part 2): 55, 1919–1920.
23. GUNDRUM, F. F. Ann. Med., 1: 420, 1920.
24. DURHAM, R. Bilateral empyema. J. A. M. A., 74: 15 (May 16) 1920.
25. GLENN, E. Empyema in children. New York State J. Med., 112: 987, 1920.
26. JEHN. 1921. Quoted by Keyes.[5]
27. LADD, W. E., and CUTLER, E. C. Empyema in children. Am. J. Dis. Child., 21: 546, 1921.
28. STENIUS, F. Abstract. J. A. M. A., November 6, 1920.
29. BECK, E. G. Bilateral empyema complicated by childbirth during influenza. Internat. Clin., 1: 27, 1921.
30. GOMEZ, RODRIGUEZ. Double empyema in a child, cured. Rev. med. de Uruguay, 24: 424 (Sept.) 1921.
31. SCHWEIZER. 1921. Quoted by Keyes.[5]
32. ANDRENELLI. 1923. Quoted by Keyes.[5]
33. ORMOS, P. Bilateral empyema. Deutsche med. Wcbnscbr., 50: 47, 1924.

34. AUER, C. Bilateral empyema. *M. J. & Rec.*, 121: 269, 1925.

35. HEDBLOM, C. A. Bilateral empyema. *J. Indiana M. A.*, 18: 209, 1925.

36. MACKAY, L. G. Bilateral empyema. *Brit. M. J.*, 1: 1124 (June) 1925.

37. GRAVES, J. Q. Double empyema of the pleural cavities. *New Orleans M. & S. J.*, 80: 420, 1928.

38. MATTHEWS, A. A. Double empyema. *S. Clin. North America*, 7: 1237, 1927.

39. SCANLAN, D. W. Bilateral empyema. *J. Med. Soc. New Jersey*, 25: 488 (July) 1928.

40. DODDS, G. H. Bilateral empyema. *J. Obst. & Gynec.*, 35: 131, 1928.

41. TIXIER and DE SEZE. Surgical treatment of bilateral pneumococcic empyema. *Arch. de méd. d'enf.*, 32: 20, 1929.

42. RAVNITSKY and BOGIN. Treatment of empyema in children. *Long Island M. J.*, 24: 191, 1930.

43. MANSON, P. *Tr. South. Surg. A.*, 42: 384, 1930.

44. MACERA, JOSE MARIE. Bilateral pleurisy—two cases. *Semana med.*, 1: 749 (March) 1931.

45. GUERIN, R. Bilateral empyema. *Bordeaux chir.*, 3: 204 (April) 1932.

46. CURTIS, G. M., and BOWMAN, H. S. Bilateral staphylococcic empyema. *S. Clin. North America*, 12: 1221 (Oct.) 1932.

47. STEINKE, C. Bilateral empyema. *J. Thoracic Surg.*, 2: 287 (Feb.) 1933.

48. MUELLER, G. P., and MOGAVERO, F. Bilateral empyema. *Ann. Surg.*, 98: 141 (July) 1933.

49. McCREA, E. D'ARCY. Bilateral empyema. *Lancet*, 1: 1117 (May 26) 1934.

50. HARRINGTON, S. W., DORSEY, J. M., and STROHL, E. L. Bilateral acute empyema. *S. Clin. North America*, 15: 1133 (Oct.) 1935.

51. STENBUCK, J. B. Case of acute purulent pericarditis with bilateral empyema treated surgically. *J. Mt. Sinai Hosp.*, 3: 149 (Nov.) 1936.

52. WILLIS, B. C. Empyema in children. *Ann. Surg.*, 107: 739 (May) 1938.

53. SNOW, W. R. Acute bilateral empyema. *Texas M. J.*, 31: 34 (May) 1935.

OVERLOOKED clinically and neglected surgically, the contusion is the most common form of cardiac trauma. The steering-wheel injury is the classic and most common method of infliction.

From—"A Textbook of Surgery" edited by Frederick Christopher (Saunders).

WOUNDS OF THE ESOPHAGUS

D. REES JENSEN, M.D.

Assistant Attending Surgeon, Knickerbocker Hospital

NEW YORK, N. Y.

WOUNDS of the esophagus are relatively uncommon, probably because, except when one is eating, the esophagus is a collapsed tube lying well protected behind the trachea and anterior to the vertebral column. This paper excludes those cases of injury resulting from the swallowing of caustic fluids, and those due to penetration of the wall by neoplasms or infection.

Injuries are usually the result of impact by a blunt or sharp object, and may be classed in two general groups, those occurring from within the tube and those from without. In the first group, the swallowing of a tack or an open pin may cause a slight injury, followed by healing without symptoms. Infrequently a sharp object such as a bone or a piece of metal may lacerate the wall, permitting the egress of food and bacteria into the cellular tissues of the neck or mediastinum, with resulting infection. More often a foreign body may lodge in the esophagus, perforating or lacerating the mucosa and causing an inflammation of the wall; with prompt removal the esophagitis rapidly subsides.

Of 205 cases of foreign bodies of various types lodged in the esophagus (reported by Jackson[1]), there were twenty-seven instances of perforation or laceration, with three deaths. In these twenty-seven, seven were in the thoracic portion. Of the three that died, the cervical portion was injured in two and the thoracic in one—all in children. Cellulitis, cervical and mediastinal abscesses and septic pneumonia were listed as the causes of death. Of those that survived the largest number were in children under 5 years, although one patient was 72 years of age.

Because of the type of infecting material and its location, a laceration entirely through the esophageal wall usually results in a cellulitis that runs a fulminating course, causing abscess formation, septic pneumonia and death. The following histories illustrate this type of injury:

CASE I. (Weir.[2]) A man of 35, while eating meat, felt a sharp object lodge in the throat. On the fourth day following a swelling developed in the right side of the neck, accompanied by pain and difficulty in swallowing. By the sixth day there was difficulty in breathing; the seventh day, extreme difficulty in swallowing and laryngeal spasms. The temperature was 102.6. Marked edema and cyanosis were present.

Operation. The deep tissues of the neck were infiltrated with pus of fetid odor with several small pus cavities present. The wound was left open and drained. Tracheotomy was later necessary. Death followed on the ninth day.

Post-Mortem Findings. A small quantity of pus was present in the anterior mediastinum. Marked edema of the right side of the epiglottis. Below the cricoid cartilage there were two openings in the esophagus. Pus was present in the intermuscular planes of both sides of the neck.

CASE II. (Osgood.[3]) A child of 11 years swallowed a small chicken bone which lodged in the throat. Late the same day soreness of the throat developed. The third day swelling was present on the right side of the neck with marked contraction of the neck muscles. The pulse was rapid and feeble. On the fourth day the same symptoms were present but more severe, with deglutition easy. The sixth day the patient had pneumonia and on the seventh day he died of suffocation.

Post-Mortem Findings. The subcutaneous tissues of the neck contained pus. Pus was present in the anterior mediastinum and in each pleural cavity. There was a left lower lobe pneumonia and a fibrinous pericarditis. On the anterior wall of the esophagus there was a long laceration. Death had resulted from suffocation.

CASE III. (Gross.[4]) A sword swallower, while performing his feat, noticed that it hurt him more than usual. He spat up blood immediately. On the third day the pain in the neck was aggravated by swallowing. The neck was swollen and tender with breathing shallow and hurried. The symptoms became more severe and on the fifth day the patient expired.

Post-Mortem Findings. There was a diffuse abscess extending along the trachea and esophagus. The mucous membrane was thickened, tough, smooth and white in color. The outermost coat and surrounding tissues were infiltrated with pus. There was slight excoriation of the upper part with no evidence of perforation or wound. The abscess had been developing prior to the last feat resulting from repeated trauma to the inner lining. The vagus nerve ran through the abscess, causing syncope and death. Edema of the tissues was not sufficient to cause death.

In another group of 628 cases of foreign bodies in the air and food passages reported by Jackson,[5] fifty-three were in the esophagus. One death occurred two years after swallowing a piece of cast iron. The patient had severe inflammation of the esophageal wall and died from bilateral pneumonia.

Apparently the esophagus can withstand most injuries to the mucosa without developing a fatal infection. With laceration of the wall permitting the passage of esophageal contents and particularly infectious agents into the surrounding loose connective tissue, an inflammatory process is initiated, and by muscular action, by gravity, and by the lymphatics, spreads rapidly along fascial planes and establishes itself in various parts of the neck, in the mediastinum, and in the lungs, causing pneumonia. Frequently there develops a suppurative pleurisy and less often a pericarditis.

Because of the nature of the pathologic process and its mode of extension, the mortality rate is very high in complete penetration of the esophageal wall from within outward. This is true in the cases where operation is delayed until the resulting abscesses become localized. Therefore incision and drainage are indicated as soon as esophageal injury can be demonstrated by the esophagoscope. This treatment provides an outlet for the accumulated infectious material and tends to avert its extension along fascial planes. It is reasonable to infer that such measures would materially lower the mortality rate, and it is relatively free from risk.

Injuries of the esophagus from without inward, particularly in the cervical portion, present a more hopeful picture. Of 172 cases that I have found reported in the literature,[6-31] there were sixty-one deaths and 110 recoveries; the result in one case was not stated.

To illustrate this type of injury and because they are reported in detail, the following four cases are selected from the above group:

CASE IV. (Nuttall.[22]) A corporal was shot through the neck June 6, 1915. The patient was received in the clearing station twenty-four hours later in collapse and with some dyspnea. He was unable to swallow and he coughed up some blood shortly after being wounded. Treatment was expectant. The third day he was improved but weak. The fifth day there was sudden severe pain in the right thorax, and on the sixth day breath sounds were absent and pleuritic effusion present on the right side. Air hunger increased, the heart failed rapidly and death followed quickly.

Post-Mortem Findings. The bullet entered the middle of the left sternocleidomastoid muscle. It passed between the esophagus and the cervical spine, perforating the posterior esophageal wall and wounded the right internal jugular vein on its posterior surface, then crossed in front of the cervical nerve trunks to come to rest on the scapula. There was a prevertebral abscess and a second larger prevertebral abscess below the former which had broken through the dome of the pleura on its inner aspect into the right pleural cavity. This contained masses of blood clot and a pint of serosanguinous fluid. The tissues of the neck were inflamed.

Whether any surgical procedure could have altered the result in the above case is uncertain. However, drainage provided by

prompt incision and exposure of the bullet tract might have averted the fatal issue.

CASE V. (Adams.[29]) In a case of an incised wound, self inflicted, the blade went through the anterior part of the neck, cutting the thyroid and cricoid cartilages, then through the trachea to the fourth ring. There were no important vessels injured. When milk was swallowed it came through the trachea into the wound. The patient was fed by enemata. On the eleventh day, the patient began taking food by mouth. The patient was discharged from the hospital cured on the forty-first day. No operative interference was necessary. The wound was allowed to heal by granulations.

CASE VI. (Longmore.[31]) A patient aged 19 years was shot through the neck and immediately spat blood. Drinking water choked him. The patient had difficulty in speaking. Twenty-five days later the patient was well except he had slight difficulty in swallowing and weakness of the voice.

CASE VII. (Blood.[30]) A Hindu woman, aged 17 years, was stabbed with a penknife causing a triangular wound just below the angle of the jaw on the right side. The direction of the wound was forward, entering the esophagus just below the thyroid cartilage. A probe passed into the wound easily excited the act of swallowing. Fluids when swallowed appeared in the wound. Treatment was expectant with no attempt at suturing. The wound healed in three days.

The following case recently seen at Knickerbocker Hospital from the surgical service of Dr. C. A. Frink, is added to the above total and reported in detail because of its interest.

A male, aged 10 years, was admitted thirty minutes after a sleighing accident. While descending a hill, he collided with the sleigh immediately in front, the steel runner of which struck him in the right side of the neck. There was an immediate hemorrhage with pain on moving the head.

General examination was negative. There was fresh blood on the posterior pharyngeal wall. At the level of the cricoid cartilage on the right side, there was a transverse laceration about 1 inch long midway between the border of the sternocleidomastoid muscle and the midline of the neck. There was no hemorrhage from the major blood vessels. Coughing and forced expiration caused air to be expelled through the neck wound. The patient was not in shock; pulse was 96, of good quality and regular.

Operation. Two and one-half hours later, under evipal and ether anesthesia, he was operated on. The sternothyroid and the omohyoid muscles were lacerated. No large blood vessels were involved. There was a longitudinal laceration ¾ inch long in the lateral wall of the hypopharynx. A duodenal tube was passed through the esophagus into the stomach without difficulty and a small piece of rubber tissue drain was inserted to the opening in the hypopharynx. The platysma and skin were closed with interrupted silk. On the second day, 20 c.c. of fluid were injected at intervals through the duodenal tube. The temperature was 100.6; pulse 115; respiration 26.

The seventh day the wound was infected, and all sutures were removed. The patient was still being fed by the duodenal tube. On the ninth day pus was draining from the wound with no involvement of the deep tissues. The patient was ambulatory but was still being fed by the tube. By the eleventh day, the patient was eating well and normally. The wound was closing by granulations. On the fourteenth day the wound was closed and the patient was discharged cured. The highest white blood count was 13,100, with 80 per cent polynuclears.

Not improbably the favorable result in this case was due to the prompt establishment of adequate drainage.

Because of their inaccessibility and ease of injuring vital structures, wounds of the esophagus in the thoracic portion are more likely to terminate fatally. In a study of 8,000 enlisted officers and men injured in battle in 1917–1918,[19] four were listed as injuries to the esophagus in this region, with three deaths. Neither the causes of death or the treatment adopted were stated.

Any attempt to suture a laceration of the esophagus is, I believe, harmful. Its structure of thin muscle lined with mucous membrane is friable tissue in which, even in the most accessible areas, it would be difficult to have the sutures hold. Furthermore, the presence of bacteria in the depths

of the wound and the extravasated blood in the loose cellular tissue provide an ideal medium for the development of an inflam-

FIG. 1. Appearance of neck wound eight months after injury.

matory process and a cellulitis which extends rapidly. The added exposure necessary to suture the esophageal wall would open up new avenues and thereby favor the spread of infection. The sound principles responsible for the evolvement of the two stage operation for diverticulum of the esophagus, lessening the probability of a fatal infection, apply equally in the treatment of these injuries. The wound should be left open, and lightly packed to permit adequate drainage; food and water should be given through an esophageal tube.

The advice of Adams eighty-five years ago, emphasizing the open treatment of such injuries, holds good today. In reporting his case, he concluded his remarks with: "Nor should it be passed unnoticed how well was exemplified in the foregoing case the propriety of avoiding plaster and sutures as is generally advised by systematic writers when treating wounds of the throat."

The frequency of cicatricial stenosis following esophageal injuries has been the subject of considerable discussion. Injuries occurring from within outward result in stenosis in a higher percentage of cases than
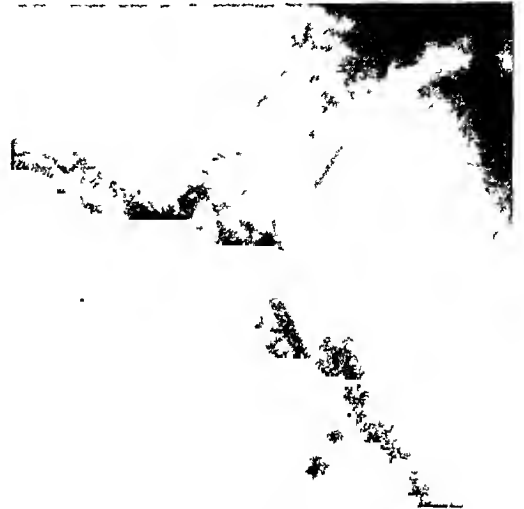
FIG. 2. X-ray of hypopharynx and esophagus. No evidence of stricture.

do those from without inward. Probably this is the result of the associated inflammation of the esophageal wall itself.

In the second group, if any of the laryngeal cartilages have been injured, even though the injury is limited to the perichondrium, there is usually a perichondritis and abscess with chondral necrosis and laryngeal stenosis. Jackson[32] states, "Chronic stenosis of the hypopharynx or of the cervical esophagus occurs in at least half of the cases."

In the case here reported a study was made eight months after injury; roentgenologic examination showed no evidence of narrowing or irregularity. (Figs. 1 and 2.)

CONCLUSIONS

1. The esophagus apparently withstands superficial injuries to a remarkable degree without developing serious infection.

2. A laceration entirely through the wall, from within outward, results in a fulminating infection of the cellular tissues of the neck and mediastinum, and, frequently, suppurative pleuritis, pneumonia and pericarditis. The mortality rate is high.

3. Because of this high mortality, early surgical drainage is indicated to avert, as

far as possible, the spreading of the infection.

4. In injuries from without inward, early establishment of drainage is indicated with no attempt at suture of the deep structures.

5. All bleeding from vessels should be promptly controlled by ligature. Feeding should be done through an esophageal tube for a period of ten days to two weeks.

6. Stenosis of the hypopharynx is a relatively common occurrence following injury.

7. Early diagnosis and early surgical drainage tend to reduce mortality and the complications which result in stenosis.

REFERENCES

1. JACKSON, C. Foreign bodies in the air and food passages. Cases #631–1155. New Bedford, 1923.
2. WEIR, R. F. Wound of the esophagus. *M. Rec.*, 21: 471, 1882.
3. OSGOOD, W. Wound of the esophagus. *Boston M. & S. J.*, 66: 165–168, 1862.
4. GROSS, G. Esophagus from a fatal case of sword swallowing. *Tr. Path. Soc.*, London, pp. 188–189, 1884.
5. JACKSON, C. Observations on the pathology of foreign bodies in the air and food passages; an analysis of 628 cases. *Surg., Gynec. & Obst.*, 28: 201–261, 1919.
6. BERGER, W. Ein Fall von Oesophagusschuss. *München. med. Wchnschr.*, 62: 1557, 1915.
7. BENOIT, G. Un cas de perforation de l'oesophage. Compliquant une plaie pénétrante de poitrine par arme à feu. *Marseille méd.*, 37: 147, 1900.
8. BERGHEIMER, S. Ueber Schussverletzungen des Halsteils des Oesophagus. Thesis, Strassburg, 1903.
9. CATHCART, C. W. Severe cut throat. *Tr. Med. Chir. Soc.*, 29: 190, 1909–1910.
10. CONTI, A. Ferita dell' esofago; flemmone periesofago; suppurazione infiltrata; esito favorevole. *Gacc. med. di Roma*, 6: 299, 1880.
11. DEBRUCK. Von einer Verletzung der Luft und Speiseröhre. *J. f. d. Chir., Arznk. u. Geburtsh.*, 1: 373–375, 1800–1801.
12. ESSER, A. Ein Fall traumatischer Oesophagusruptur. *Deutsche Ztschr. f. d. ges. gerichtl. Med.*, 9: 312–316, 1927.
13. FRANÇOIS, J. Contribution a l'etude des plaies de l'oesophage. Thesis, Nancy, 1884.
14. HUTTON, E. Wound of the esophagus, etc. *Proc. Path. Doc., Dublin*, p. 116, 1854–1855.
15. LOMBARD, P. Plaiede l'oesophage cervical par balle; mediastinite gangreneuse; pleurisie et péricardite purulentes. *Bull. et mém. Soc. anat. de Paris*, 91: 916, 9116.
16. MADELUNG. Einige Kriegsverletzungen des Oesophagus. *Deutsche med. Wchnschr.*, 41: 124–127, 1915.
17. MANCINI, F. Sutura dell' esofago e della laringe per grave ferimento d'arma perforante e bitagliente. *Raccoglitore med., Forli*, 22: 57–62, 1896.
18. MARCHETTI, G. Con tributo alla cura delle ferite da taglio interessanti trasversalmente la regione anteriore del colle ed accompagnate a lesioni della trachea e dell' esofago. *Gazz. d'osp., Milano*, 40: 746, 1919.
19. Medical Department of the U. S. Army. Part i, Vol. ii, p. 68.
20. NUTTALL, A. W. Bullet wound of the esophagus. *Lancet*, p. 1350, 1915.
21. OLIVER, J. P. Pistol shot wound of the neck; esophagus and larynx wounded; tracheotomy thirty hours afterwards; recovery. *Tr. Texas M. A.*, 23: 183–186, 1891.
22. PELOUX, A. Des plaies de l'oesophage. Thesis, Montpellier, 1902.
23. RIDENOUR, A. W. Injury to esophagus. *Toledo M. & S. J.*, 4: 17, 1880.
24. SAFRANEK, J. Durch traumatische Verletzung entstandener grosser Defekt des Larynx und des Oesophagus. *Ztschr. f. Hals.-, Nasen- u. Ohrenh.*, 3: 561, 1911.
25. SCOTT, G. D. Extensive cut throat with complete laceration of trachea and esophagus. *J. A. M. A.*, 90: 629, 1928.
26. SÜSSKAND. Ueber die gerichtsärztliche Beurtheilung der Verletzungen der Speiseröhre. *Vrtljschr. f. gerichtl. Med.*, 10: Suppl., 1–27, 1895.
27. TENANI, O. Ferita d'arma da taglio trasversale al collo, interessante la trachea e l'esofago; sutura della trachea e dell'esofago; trachcotomia; guarigione. *Gazz. d. osp., Milano*, 35: 1249–1251, 1914.
28. ZELLER, O. Zur Behandlung der Speiseröhrenverletzungen am Halse. *München. med. Wchnschr.*, 916, 1916.
29. ADAMS, J. Self inflicted wound of the throat, laying open oesophagus; recovery. *Lancet*, 1: 699, 1850.
30. BLOOD, J. Case of wound of the esophagus. *Ind. Med. Gaz.*, 18: 101, 1883.
31. LONGMORE, T. Gunshot wound of the neck, wound of the esophagus; rapid recovery. *Lancet*, 2: 436, 1855.
32. JACKSON, C. Personal communication.

THE FALLIBILITY OF PROLAN A EXCRETION AS A PROGNOSTIC AGENT IN CASES OF TERATOMA TESTIS

EDWIN J. GRACE, M.D., F.A.C.S.

Attending Surgeon, Kings County, Kingston Avenue and St. Mary's Hospitals

BROOKLYN, NEW YORK

DURING the last decade biochemical research on the sex hormones has added materially to our clinical knowledge. At the same time clinical observations have led to further study. Since Zondek first noted the appearance of the sex hormone of the anterior hypophysis, prolan A, in the urine of a man suffering from teratoma testis, numerous observers in many countries have made similar observations, and Ferguson has pointed out that the appearance of this hormone, which is not normally present in the urine, constitutes a valuable diagnostic agent. The writer's experience confirms this point of view. Prolan A is not present in the urine of healthy men, nor in that of patients with benign lesions of the testis; but it is consistently present in those with teratoma testis.

Its value as a prognostic agent is less certain. The case cited here is reported with the hope that it may throw further light on this questionable point. In 1934 Ferguson[1] reported the effects of irradiation treatment on the prolan A output. He noted that if the excretion of the hormone was unaffected by irradiation, the prognosis was bad. In all such instances, the patients died. If, however, irradiation treatment was followed by a drop in the excretion of prolan A in the urine, Ferguson considered the rapidity and extent of this drop "a good index of the radiosensitivity of the tumor, and a *reliable* factor on which to base the prognosis." (The italics are ours.) He further observes that in all of the recurrent cases in his series, "an increase in the excretion of prolan A in the urine was observed anywhere from two weeks to three months before the metastatic lesions became clinically demonstrable."

Our experience with the following case in which surgical removal of the tumor was followed by a drop in prolan A output from 719 units to 450 units does not lead us to believe that prolan A output is a "reliable" factor on which to base prognosis.

The patient was a male, aged 33 years. His past history was negative except that his aunt had died of cancer of the breast. In March, 1934, during an examination for diabetes mellitus and overweight, the patient remarked that he thought the right testicle was getting larger. The patient was overweight about 80 pounds (weight 240 pounds). The testicle was hard, smooth, symmetrical, and not painful. It seemed probable that if any tumor was present, it was benign; but nevertheless a consultation was held by Drs. Ewing, Ferguson, and the writer. An aspiration biopsy was advised and the condition proved to be teratoma, grade 4, of the right testicle.

X-ray therapy to the testicle, abdomen, and chest was started immediately. Three months later, June 6, 1934, the tumor had diminished greatly in size and it seemed probable that all the viable cells in the tumor had been destroyed. The testicle and cord with all its contents were removed surgically. Pathologic studies of the tissue from the cord and primary tumor showed no viable tumor cells.

Estimates of the prolan A excretion in the urine were begun on April 30, 1934, one month after recognition of the tumor. At this time there were 2,000 units; three days later the output had dropped to 250 units under irradiation treatment, and we became hopeful of a good prognosis, basing our judgment on the rapid drop. On May 14, there was a rise to 1,000 units, but after eight more days, the output had again dropped to 200 units. Operation was performed on June 6. On the 14th, the output was 350 units, but as Ferguson had stated that an output of 400 mouse units or less was consistent with clinical cure, we were

still hopeful. *X*-ray examination of the chest was negative, and by April 8, 1935 the prolan A output had dropped to 100 units. Following this, there was a gradual rise in output to 400 units in September and 500 in December. On December 23, 1935 the patient developed a cough with hemorrhage. The *x*-ray showed a large, single, metastatic nodule at the right base, just above the diaphragm. *X*-ray therapy was given to this nodule, and the prolan A output again dropped rapidly, reaching 100 units in April, 1936. But the patient's condition grew steadily worse. He expired on October 31, 1936. The last prolan A reading was 500 units on July 27, 1936.

This case is interesting because of the high degree of fluctuation in the prolan A output, with the rapid declines to as low as 100 units, giving us hope that the patient would escape metastasis and recover. This may be regarded as an exceptional case. However, as we have no way of knowing when we are dealing with an exceptional case, it seems inadvisable to place too great reliance on prolan A output as a prognostic agent. Although there was a gradual rise in output just before the appearance of metastasis, this was not so marked as Ferguson's observations would have led us to believe.

REFERENCE

1. Ferguson, R. *J. Urol.*, March, 1934.

Hormones do not act in vitro on inanimate matter. Their metabolic effects are obtained only by contact with living cells.
From—"The Endocrine Glands" by Max A. Goldzieher (D. Appleton-Century).

TUBERCULOUS MENINGITIS FOLLOWING NEPHRECTOMY FOR RENAL TUBERCULOSIS*†

THREE CASE REPORTS

G. A. HAWES, M.D.

NEW YORK CITY

ALTHOUGH there have been quite a number of cases of tuberculous meningitis following nephrectomy for renal tuberculosis reported in the literature, yet this sequel is of sufficient rarity to justify recording these cases in detail. In searching through the cases of the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital, only three such records have been found. It is most likely that the meningitis would have developed in these cases, irrespective of nephrectomy, as a result of tuberculous lesions elsewhere in the body. It should be stressed that, in the local treatment of renal tuberculosis, one should be guided by the general physical condition of the entire body with reference to tuberculous lesions existing in other organs before local measures are instituted.

CASE I. C. E., age 23, single, was admitted to the Department of Urology (James Buchanan Brady Foundation) on October 15, 1935, complaining of frequency, urgency, burning, and nocturia. Approximately one year before admission he was accidentally struck on his right testicle, which soon became swollen and painful. The pain subsided within a week, but the swelling persisted. Two months after this accident he began to have burning on urination, urgency, frequency and nocturia, followed within two months by a painless hematuria associated with the passage of gross particles of "flesh." Five months after the testicle became swollen a right orchidectomy was performed in another hospital, which returned a pathologic report of tuberculous epididymitis. The bladder symptoms of frequency, urgency, burning, and nocturia gradually became exaggerated, so that he was voiding every half hour during the day, and with considerable discomfort.

The patient had enjoyed good health until his present illness began. He had bronchopneumonia without sequelae at the age of 10 years and rheumatic fever without sequelae as a child. The only operation he had experienced was the right orchidectomy, seven months before admission.

Family history was irrelevant, except that one paternal aunt had tuberculosis, but the patient had not been exposed.

Physical Examination. The patient was a fairly well developed and well nourished white male of 23 years, in no acute distress, alert and coöperative. There was no local or general glandular enlargement. Examination of the head, neck, and chest revealed no pathologic changes except dental caries, and granular breath sounds, but no râles. The right kidney was easily palpable, but not tender. The penis was normal; the right testicle absent; the left epididymis was indurated and enlarged at the globus major. The prostate was normal in size, elastic in consistency, non-tender, and the seminal vesicles were not palpable. Reflexes were present and active.

The urine was clear and acid, showed a trace of albumin, but was negative for sugar. An occasional clump of leucocytes but no red blood cells were demonstrable. The blood Wassermann was negative. Blood urea was 8 mg. per 100 c.c., and the blood sugar 111 mg. Hemoglobin was 90 per cent; red blood cells 4,800,-000; white cells 8,000 with 63 per cent polys. The sediment from centrifuged urine, stained with Ziehl-Neelsen stain, was positive for acid-fast bacilli and the guinea pig inoculation test was positive.

Cystoscopy was done under spinal anesthesia, the No. 24 F. Brown Buerger cystoscope passing without difficulty. The vesical fundus showed generalized edema with an unusual amount of edema and injection in the region of the right ureteral orifice. The left ureteral orifice was also edematous and injected, but not so markedly as the right. The vesical orifice was granular and bled easily upon manipulation of the cystoscope. A No. 5 F. lead catheter passed to the kidney pelvis on the right side with great difficulty, while one of this caliber passed to the left kidney pelvis with ease. Specimens collected from the kidney pelves showed diminished function of the right kidney.

X-rays showed the kidney shadows to be normal in size and in good position. There were no areas of calcification in the urinary tract. The right pyelogram showed shaggy, distorted and distended middle and lower calyces, while the upper calyx was normal. The right ureterogram had a beaded appearance indicative of disease in the upper portion of this organ. The left pyeloureterogram showed a bifid type of kidney pelvis, with normal calyces. The left ureter presented no pathologic changes. A diagnosis of tuberculosis of the lower pole of the right kidney and ureter was made. (Fig. 1.)

X-ray of the chest did not suggest recent pulmonary involvement.

Operation. On October 18, 1935, a right nephrectomy was done by Dr. O. S. Lowsley under spinal anesthesia, using 200 mg. of novocaine crystals. The lower pole of the kidney was densely adherent, but was delivered into the wound without difficulty. The ureter was dissected down as low as possible, ligated and severed, phenol and glycerin being used to cauterize the stump. A kidney pedicle clamp was placed on the pedicle, and two No. 2 ligatures were placed below this. The kidney was then cut off above the pedicle clamp, leaving a good rosette of tissue, and following this a transfixation suture was placed through the renal pedicle. The wound was repaired in the usual manner, with one Penrose drain. The patient left the operating room in excellent condition.

Convalescence. For the first six days postoperatively the temperature ranged between 102 and 104 degrees, with a pulse from 90 to 100 per minute. The wound drained a moderate amount of purulent material. There was some abdominal distention which disappeared on the fifth postoperative day. His fluid intake was fairly satisfactory and there was no evidence of

Fig. 1. Case 1. Note shaggy, moth-eaten, distorted and distended middle and lower calyces on the right side. Also the right ureter is slightly dilated with a beaded appearance. The left pyeloureterogram is normal.

deficiency of the left kidney. At no time during his postoperative hospital convalescence did he complain of headache.

The patient was discharged on November 10, 1935, the twenty-third postoperative day, feeling quite well except for the symptoms of cystitis.

The pathologist described the kidney as follows: The upper two-thirds of the kidney were grossly normal in appearance except for a slight dilatation of the pelvis. The lower third was partially destroyed by an ulcerative process involving the calyces, and also decreased in size due to a depressed scar of the lateral aspect. The cavities were lined by soft yellowish-white tissue, in places distinctly caseous in appearance. The microscopic examination was typical of renal tuberculosis, showing epithelioid cells and tubercles.

Exactly forty days after dismissal from the hospital, December 20, 1935, the patient was readmitted with the following history. After returning home he remained on a limited routine with rest and gradually regained

strength. Approximately two weeks before this admission he developed a "cold" in his head and chest, accompanied by chills, fever, and dull, aching pains in his legs. With the continuance of the chills and fever, the pain seemed to progress upward in his body and spine to his shoulders, neck, and head. At the time of admission he complained of severe pain and stiffness of his neck. Nervousness was a prominent feature, requiring sedatives for rest.

The positive physical findings at this time were: slight strabismus of left eye, discharge from right ear drum, stiffness of the neck, well healed right nephrectomy scar, a positive Kernig, and hyperactive reflexes. The white cell count was 10,000, with 79 per cent polys.

On December 22, 1935, 65 days after the nephrectomy, a spinal puncture was done. The spinal fluid was under considerable pressure and cloudy, and the cell count was 509 (lymphocytes), but no tubercle bacilli or other bacteria could be demonstrated on smears. A culture, however, was positive for tubercle bacilli after thirty-five days. The temperature fluctuated between 99 and 101 degrees. Frequent spinal punctures were done to relieve the pressure and he was treated with old tuberculin. He gradually became mentally confused, irrational, comatose, and died on January 10, 1936, twenty-one days after the second admission and eighty-four days postoperatively.

A complete autopsy was obtained, and the following diagnoses were made: (1) tuberculosis of stump of right ureter; (2) tuberculosis of urinary bladder, miliary tubercles of spleen and liver, calcified tuberculous nodules of bronchial lymph node on the left, and bilateral otitis media. The neurologic examination revealed a marked meningeal exudate covering the base of the brain with a lesser amount of exudate over the hemispheres. Marked congestion of the cortical veins was present. There was a beginning hydrocephalus of the lateral ventricles from obstruction by the exudate at the base of the brain. The microscopic picture was one of tuberculous meningitis.

CASE II. Mrs. M. M., age 43, was admitted to the Department of Urology, James Buchanan Brady Foundation, on December 6, 1936, complaining of pus in her urine. Approximately two months before admission she noticed that her urine was cloudy. She had no dysuria, burning, frequency, urgency, nocturia, or hematuria. She had her urine examined and it showed numerous pus cells. Two years before admission had had an attack of dysuria, frequency, and nocturia which lasted several weeks, and then subsided after oral medication. Three months before admission she reported to her local physician because of loss of weight and a feeling of malaise. Since she failed to improve she was referred for further study.

The patient had developed pleurisy ten years before, and one year later a diagnosis of pulmonary tuberculosis was made. She was treated by rest, diet, sunshine, etc., for a period of one year, and was told she was cured. Up to three months before the present admission she had felt quite well, with no clinical symptoms of pulmonary tuberculosis.

Family history was entirely irrelevant.

Physical examination revealed a fairly well developed and nourished middle aged female in no acute distress. The head, neck, and chest showed no pathologic changes, there being no râles or abnormal dull areas present. The abdomen was normal, the lower pole of the left kidney being palpable but not tender. The rest of the examination was normal, except for slightly hyperactive reflexes.

Urinalysis showed the urine to be cloudy, acid, with a specific gravity of 1.020, a trace of albumin, no sugar, 5 to 10 red blood cells and numerous pus cells to the high power field. Urinary sediment stained with carbolfuchsin showed a moderate number of acid-fast bacilli. Hemoglobin was 78 per cent, and white blood cells 8,800. The Wassermann was negative. The blood urea was 10 mg., and the blood sugar 90 mg. per 100 c.c. of blood.

Cystoscopy and Pyelograms. The No. 24 F. Brown Buerger cystoscope passed easily. The bladder showed generalized cystitis with marked trigonitis. The left ureteral orifice could not be visualized owing to inflammation and edema, and the right ureteral orifice was slightly injected. A No. 6 F. catheter was passed to the left kidney pelvis without obstruction, but a similar catheter passed only one half the distance to the right kidney pelvis, meeting an obstruction at this point. Specimens collected showed a diminution in function of the left kidney, the phenolsulphonphthalein appearing in ten minutes from the left side and 4 per cent being excreted in ten minutes, as compared with an appearance in five minutes with 8 per cent excreted in ten minutes on the right side. There were also 50 red and 50 white cells to

the high power field from the left pelvis, while the urine from the right kidney was free of pus and blood.

X-Rays. The plain plate showed both kidney shadows normal in size and in good position. Near the tip of the left catheter was a shadow approximately 7 mm. in diameter which probably represented a calculus. No other shadows indicative of calculi were noted in the urinary tract. The right pyelogram indicated that the pelvis was bifid in type but within normal limits. The right ureter filled incompletely, but no evidence of pathologic change was noted. The left pyelogram showed the pelvis of normal size, but revealed an absence of the superior and inferior major calyces; those that were present were slightly dilated and blunted. The left ureter filled well throughout, with a suggestion of a slight angulation at the ureteropelvic junction. The remainder of the ureter was slightly dilated and tortuous. A diagnosis of left renal tuberculosis was made and the patient was advised to have a left nephrectomy. (Fig. 2.)

Operation. On December 7, 1936, a left nephrectomy was performed under spinal anesthesia, using 150 mg. of novocaine crystals. The kidney was easily freed from the surrounding fat and removed without difficulty with the same technique as that described in the first case. The patient left the operating room in excellent condition.

Convalescence was entirely uneventful, the temperature not going above 101°F. The patient was discharged on the eighteenth postoperative day, December 25, 1936, with the wound completely healed.

Pathologic Report. The kidney was usual in size, but the capsule stripped with some difficulty in a few areas. There were a few demonstrable tubercles on the surface. On serial section, there were many areas of cheesy granular material, confined mainly to the upper and lower poles. Several of these caseous areas had ulcerated through into the minor calyces. The kidney pelvis was slightly dilated and the mucosal lining thickened with several minute yellow nodules. The left ureter was not abnormal. Microscopic examination revealed many tubercles, with the typical Langhans giant cells.

Three weeks after discharge, or thirty-nine days after operation, she was readmitted to the hospital complaining of frontal headaches,

stiffness of the neck and generalized body pains of four days' duration.

She also complained of burning, frequency,

Fig. 2. Case II. Note absence of superior and inferior calyces, while those that are present are slightly dilated and blunted on the left side. The left ureter is slightly dilated and tortuous. The right pyelogram shows a bifid pelvis.

nocturia, and dysuria. She was nervous, had a stiff neck, dulness and scattered râles at the apex of the right lung anteriorly and posteriorly. A well-healed left nephrectomy scar, and hyperactive reflexes completed the picture. Her temperature was 100.2°F., pulse 90, and respirations 26.

On the day of admission a lumbar puncture was done, removing 15 c.c. of slightly cloudy spinal fluid, under increased pressure. The cell count was 500 white corpuscles, 10 per cent of which were lymphocytes, but no tubercle bacilli could be demonstrated. The sugar content was decreased while the protein was increased. Blood examination showed a hemoglobin of 88 per cent, red cells 4,360,000, leucocytes 13,500, polys 84 per cent, lymphocytes 4 per cent, monocytes 12 per cent. X-ray of the chest demonstrated an old healed tuberculous process in both the upper lobes.

A second lumbar puncture was performed two days after this admission, and the centrifugalized sediment, stained with carbol-fuchsin, contained acid-fast bacilli. A guinea

pig inoculation with the spinal fluid was reported positive for tubercle bacilli six weeks later.

FIG. 3. Case III. The left pyelogram demonstrates a dilated pelvis, the upper calyx being defective in contour with some shagginess, and the middle and lower calyces irregular in outline. The left ureter is dilated and moth-eaten in appearance.

During the two weeks of the second admission, the patient gradually grew progressively worse, having severe headaches, vomiting, rigidity of her neck. She was semicomatose, finally lapsing into coma and expiring on the fifteenth day. After a microscopic diagnosis of tuberculous meningitis was established, treatment consisted of frequent lumbar punctures, sedatives, and nursing care. An autopsy could not be obtained; however, there was no question as to the final diagnosis, since the tubercle bacilli were demonstrated in the spinal fluid as well as by a positive guinea pig inoculation test.

CASE III. Mr. F. R., age 28, single, was admitted to the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital on September 29, 1937 complaining of pus and blood in his urine of one month's duration. Associated with these symptoms were slight burning on urination, frequency, and nocturia. Three days before admission he began to have a dull aching pain in the region of his right kidney, associated with an elevation of

temperature. His past and family history were irrelevant.

Temperature was 103°F., pulse 108, blood pressure 120/80. The patient was well developed and nourished, but acutely ill. His skin was hot and moist, his lungs showed decreased resonance, bronchial breath sounds, and a few fine râles in the left apex, posteriorly. The heart was normal. The abdomen showed no abnormal masses or tender areas. The genitalia normal and the reflexes present and active.

The urine had a trace of albumin, numerous red blood cells and 1 to 3 leucocytes to the high power field. Urine from the left kidney contained acid-fast bacilli. Hemoglobin was 88 per cent, white count 6,000, blood Wassermann negative. Blood urea was 13 and blood sugar 120 mg. per 100 c.c. of blood. The P.S.P. test gave 96 per cent in three hours. A tryptophane test on the spinal fluid was negative.

Plain x-ray plate of the chest showed fairly soft bilateral apical infiltration which suggested recent disease. Cystoscopy indicated good function from both kidneys, but a plain x-ray of the abdomen showed a small calculus in the right kidney substance. The left pyelogram showed the upper calyx defective in contour, with some shagginess, and the middle and lower calyces were irregular in outline. The renal pelvis was dilated, and the ureter moderately dilated and moth eaten in appearance. The right pyeloureterogram was normal. Uroselectan series confirmed the retrograde findings. (Fig. 3.)

On October 4, 1937 under spinal anesthesia, a left nephrectomy was done with removal of as much of the ureter as possible. On the day after operation the temperature rose to 104 degrees, and then dropped to 101 on the second postoperative day, gradually returning to normal at the end of the first week. The postoperative convalescence was uneventful except for a slight elevation of temperature and a left frontal headache on the twentieth day, at which time the patient was discharged. A small draining sinus remained in the posterior angle of the nephrectomy wound.

The pathologic report of the kidney and ureter showed renal and ureteral tuberculosis.

The patient was readmitted to the hospital five days after discharge, complaining of a severe frontal headache which had persisted since he had left the hospital. He also complained of slight stiffness of his neck. Except for

this, physical examination at this time was negative. The knee jerks were equal and active, not exaggerated, and Kernig's sign was negative. The blood pressure was 135/85. The left kidney incision was well healed.

A lumbar puncture on the day of admission showed the spinal fluid under increased pressure, clear in color, with a cell count of 55, but with no acid-fast organisms. The tryptophane test was negative. On the second day a bilateral positive Kernig's sign was noted. During the next two weeks, four lumbar punctures were done, which relieved the headache only temporarily. The cell count of each specimen of spinal fluid increased, the last cell count being 320, with 72 per cent lymphocytes. The tryptophane test of the third specimen of spinal fluid was slightly positive for tuberculous meningitis. The patient gradually became moribund and expired on the seventeenth day of the second admission, forty-two days after nephrectomy.

The following diagnoses were made from the autopsy: (1) chronic pulmonary tuberculosis involving the upper fourth of the right upper lobe and upper third of left upper lobe, with fibrosis, caseous and calcified nodules; (2) healed nephrectomy wound (left); (3) acute disseminated miliary tuberculosis with miliary tubercles of all lobes of lungs, tuberculous

meningitis (smears made from the meninges showed large numbers of acid-fast organisms), and miliary tubercles in spleen and liver; (4) tuberculosis of left ureter, prostate and right seminal vesicle; (5) chronic cystitis with multiple tuberculous erosions of mucosa; (6) extensive bilateral fibrous pleural adhesions.

CONCLUSIONS

In view of the fact that tuberculous meningitis can follow nephrectomy for renal tuberculosis, one should give a guarded prognosis when patients present symptoms of meningeal irritation after such an operative procedure.

REFERENCES

1. TURNER, J. H. Tuberculous meningitis following nephrectomy. *J. Urol.*, 34: 216–221 (Sept.) 1935.
2. FULLERTON, A. Statistics of postoperative survival in renal tuberculosis. *Brit. M. J.*, 2: 630–633 (Oct. 8) 1927.
3. ALEXANDER, R. C. Series of nephrectomies for tuberculosis. *Edinburgh M. J.*, 45: 127–131 (March) 1936.
4. WOODRUFF, S. R., and BUMPUS, H. C., JR. Is nephrectomy always indicated following a diagnosis of unilateral renal tuberculosis? *J. A. M. A.*, 104: 716–720 (March 2) 1935.

POSTOPERATIVE CYSTITIS*

Frank Coleman Hamm, m.d., f.a.c.s.

Attending Urologist, St. Mary's and Bushwick Hospitals; Associate Urologist, Brooklyn Hospital

BROOKLYN, NEW YORK

PERHAPS a more fitting title for this paper would be "Postoperative Infections of the Urinary Tract," inasmuch as infection of the bladder by itself does not usually occur.

The diagnosis of "cystitis" should be made advisedly. It is justified only after complete study of the entire urinary apparatus. The clinician's attention is usually drawn to the urinary tract by symptoms arising from the bladder, which may for this reason be regarded as the spokesman for the rest of the urinary system. Inflammatory processes of the kidney, pelvis and ureter have no way of manifesting themselves clinically unless the process is so severe that urinary obstruction occurs, with dilatation of the renal pelvis, or if the inflammatory process involves the capsule of the kidney. These last mentioned conditions will produce renal pain.

The bladder, under normal circumstance, is highly resistant to infection. This statement is supported by abundant evidence, both experimental and clinical. Pathogenic bacteria in large numbers may be introduced into a normal bladder without serious consequence. Infection is seen infrequently even when the introduction of bacteria is accompanied by moderate trauma, as seen following the inexpert or heavy-handed use of the cystoscope. If the above statement were untrue it would be almost impossible to carry on cystoscopy or many of the operative cystoscopic procedures.

Severe postoperative infections are the source of great annoyance to the patient as well as to the surgeon. This complication may prolong the period of convalescence and the distress to the patient may be greater than the operation itself. Fortunately most of the severe types may be either prevented or rapidly cleared up under proper management.

A careful study of postoperative urine specimens will show the presence of leucocytes in a surprising number of cases. Evidence of infection varies from the mildest group, in which pus cells or bacteria are found for a brief period, to the more severe type where infection is evidenced by pus and bacteria in the urine in large quantities and clinical symptoms of chills and fever, dysuria and frequency. In cases of severe systemic reaction as evidenced by chills, high fever and tenderness in the costovertebral areas an infection of the entire urinary tract may be assumed.

Many of the above-mentioned are only laboratory infections, others may be more serious. If treatment is promptly instituted during their incipiency the severe forms may be avoided.

By doing postoperative urine examinations routinely the cause of an otherwise unexplained fever during convalescence may be discovered.

We have pointed out that the bladder may be counted on to stand a great deal of abuse. What then may be considered the predisposing factors in producing the more severe type of postoperative cystitis? Postoperative retention of urine with severe overdistention is the principal factor. The overdistended bladder affords all the necessary prerequisites for infection.

Postoperative distention is produced by an interruption of the reflex arc between the spinal cord and the bladder. This reflex retention occurs following a variety of surgical procedures, more commonly those in the lower part of the abdomen, the inguinal and perineal regions and also after gynecologic surgery and normal labor. It is

---

* Read before the Surgical Section of the New York Academy of Medicine, April 1, 1938.

probable that the reflex arc is so occupied by the transmission of the pain impulse that the voiding stimulus is unable to make itself felt.

The bladder is a hollow muscular organ lined on the inner side by mucous membrane. On its outer surface large tortuous blood vessels are seen. The wall is capable of a certain degree of stretching and contraction and to that reasonable degree the blood supply has become accustomed. The normal capacity of the bladder is a variable factor, and no doubt many persons may be gradually trained greatly to increase their bladder capacity. If, however, a sudden severe distention occurs there is a stretching of the whole bladder wall which results in a disturbance of its blood supply. The bladder is capable of distention to an enormous degree without rupture; in fact, rupture of the distended bladder from distention alone is probably unknown.

During the process of distention the thin-walled veins become compressed, the arteries are over-expanded by the positive force of the blood pressure. Following the release of this pressure marked congestion of the mucous membrane of the bladder may occur. Microscopically the picture is one of congestion, edema and petechial hemorrhage. More severe cases show gross bleeding, and the bladder is then properly prepared for infection. This condition has been commonly referred to as a "catheter cystitis." Many patients have been allowed to endure the discomfort of acute retention because the surgeon feared the catheter. If the patient is not permitted to become distended there is no danger in its use. Infection will frequently occur in this damaged mucosa without the introduction of the catheter.

One occasionally hears the remark that the surgeon is reluctant to start catheterizing a patient because he will be obliged to continue it; in other words, that catheterization will delay the resumption of the normal voiding reflex. This is obviously untrue. Certain patients will be delayed for longer periods in reëstablishing their void-

ing reflex than others, but if the bladder musculature is stretched beyond a certain point it is incapable of forceful contraction and the bladder must be put at rest in order that its musculature may regain its tone.

The management of these cases may be divided into prevention and treatment. Under prevention the most important point is the avoidance of postoperative distention. The question that next occurs is how long shall we let the patient go before using the catheter? Many factors influence the secretion of urine. General anesthetics, particularly ether and nitrous oxide, inhibit its secretion. However, nausea may follow anesthesias so that no fluids will be taken for several hours after. It is difficult to lay down a specific law that will apply to all cases. It is possible to assume however, that normal kidneys will secrete on the average about an ounce of urine per hour. Generally speaking catheterization should be done about twelve hours after operation if the patient has not voided.

If catheterization has been delayed and a large amount of urine has accumulated, it is safer to withdraw only about 12 ounces at a time. The same amount may be withdrawn at two hour intervals until the bladder is emptied. Many observers believe that sudden withdrawal of the entire contents of the bladder adds to the congestion and submucosal hemorrhage. The subject of slow decompression of the bladder is being questioned in certain quarters but so far there are not sufficient data to warrant discontinuing it.

In cases where prolonged postoperative distention has occurred and there is no tendency on the part of the bladder musculature to contract, an indwelling catheter may be employed to advantage. This permits the muscular wall to remain at rest and hastens its recovery. Such indwelling catheters require considerable attention to see that they are draining properly. An obstructed catheter is worse than no catheter at all. The bladder should be washed out thoroughly once or twice a day with a

mild antiseptic solution under strictly aseptic technique.

Several points should be emphasized in connection with the technique of catheterization. This is such a common procedure that one would assume that all the careful detail had been worked out to insure the comfort of the patient. Unfortunately this is not the case. One of the most effective methods of making catheterization less unpleasant is the use of a small catheter; a size 14 F or 16 F is ample. In the female, rigid or semi-rigid catheters of this size are particularly convenient. They may be obtained in woven silk or in a new composition that may be boiled. In the male, a fairly new, soft rubber catheter of the same size is preferred. Old rubber catheters that have been boiled many times lose their firmness and the smooth external covering becomes roughened, making its use difficult. The external meatus is gently sponged with an antiseptic solution. Ample lubrication should be used. (I recently visited a hospital where the use of a lubricant on the catheter was forbidden by the training school.)

It is wise then to place postoperative cases on an eight hour catheterizing schedule if they show no tendency to void in the first ten to twelve hours. As soon as the mechanical difficulties of retention have been taken care of we should further insure ourselves against infection by giving a urinary antiseptic. Fortunately at the present time we have two very efficient drugs for this purpose, namely, ammonium mandelate and sulfanilamide. Ammonium mandelate is probably the less toxic of the two, and it is highly efficient.

Mandelic acid compounds are a direct outgrowth of the use of the ketogenic diet. This diet was too involved to be applied to ambulatory patients, so a search was instituted to find an organic acid that could be taken orally and would be excreted in the urine unchanged. The organic acid found in the almond was the result. It is irritating to the gastric mucosa and its administration has caused considerable gastric distress. Recently it has been put up in enteric-coated tablets. This preparation seems to be very satisfactory.

The bacteriostatic effect of mandelic acid compounds is produced by increasing the acidity of the urine. A hydrogen ion concentration of 5.5 to 5 must be obtained for the best results. The drug should be given in adequate dosage, 4 to 8 gm. a day. It is most efficient when used against the colon bacillus group of organisms which are at fault in about 70 per cent of all urinary infections. Almost all the cases under discussion in this paper fall in this category, with one outstanding exception, the proteus ammoniae or urea-splitting group of organisms. Ammonium mandelate not only does not help these cases, but makes them worse. The ammonium-splitting organisms attack the ammonium mandelate and split off the ammonia; instead of obtaining a higher degree of acidity the opposite is accomplished, and the free ammonia makes the urine less acid.

Administration of mandelate compounds should not be continued in large doses for a period longer than ten days. Prolonged administration is irritating to the kidney and diminished renal function may be demonstrated.

Certain patients are sensitized to mandelic acid. In one case under the author's observation recently, severe edema of the eyelids and lips and ulcerations of the mouth and tongue occurred. Tinnitus and temporary deafness have also been observed. These symptoms clear up rapidly when the drug is discontinued.

Striking results will usually be observed within seventy-two hours. If this does not occur the suspicions of the clinician should become aroused and other studies may be indicated.

Sulfanilamide is an excellent urinary antiseptic and is extremely efficient in doses of as little as 40 gr. a day. The action of sulfanilamide is not definitely established, but it is excreted largely in the urine and is effective when used for all

common bacteria found in the urine with the exception of the staphylococcus.

In contradistinction to the mandelic acid group of antiseptics, the efficiency of sulfanilamide is enhanced by reducing the acidity of the urine. This is readily accomplished by the administration of one dram of bicarbonate of soda or some similar alkali twice daily.

Ordinarily, few if any untoward symptoms will be observed when sulfanilamide is administered in 10 gr. doses, four times daily, for a period of less than ten days. The toxic symptoms produced by this drug occur following the administration of much larger doses.

No urinary antiseptic should be continued for a period of more than ten days. If the urine is not clear by this time adequate investigation is indicated.

If the above-mentioned recommendations are carried out, most of the difficulties may be prevented, although occasionally infections occur in spite of the very best care. The treatment of the well-developed case is in principle the same as that outlined under prevention: catheterize the patient after voiding to be sure he is emptying his bladder. Daily bladder lavage is indicated. Any mild antiseptic solution, for example warm boric solution, may be used for this purpose.

Urinary antiseptics are employed as previously mentioned. Fluid intake is limited to about 2,000 c.c. for each twenty-four hours, so that the antiseptic will not be excessively diluted in the urine. In patients with high fever it is unnecessary to restrict fluids. In certain specialized types of surgery adequate provision for the urinary drainage should be made at the outset. This is particularly true in radical operations on the large bowel where there is considerable disturbance of pelvic structures and injury to the nerve supply to the bladder. Close cooperation between the urologist and the surgeon should be maintained in these cases.

The advent of tidal drainage has been of great benefit in the management of all types of cord bladder during the period of hospitalization. It consists of an indwelling catheter connected with a closed system of irrigation. The irrigating system is equipped with automatic regulators that allow the bladder to be filled and emptied at regular intervals. The rate of filling and emptying may be regulated to almost any speed. Operations on the spinal cord and cauda equina also fall into this group. With this device it is possible to keep such cases free of infection for an indefinite period.

Obstructive lesions should be watched for, particularly in men past 60 years of age. Enlargement of the prostate gland begins to appear at about this time. The patient may be completely unaware that any enlargement of the prostate exists, but following operation, when the voiding stimulus is interfered with, and perhaps the general muscular tone diminished, he finds himself unable to void even after a prolonged period of catheterization. Surgical interference for the relief of obstruction may be necessary.

Another group of postoperative urinary infections does not, strictly speaking, come under the heading of postoperative cystitis, but is mentioned here briefly. This group is composed of those who are obliged to spend a long period of time in bed, following operation, as for example in chronic osteomyelitis, empyema, certain fractures and dislocations immobilized for long periods of time in traction devices, frames, etc.

Any patient confined to bed or immobilized for a long period of time should have regular and careful urine examinations. The urinary tract drains properly only when we are in the erect, normal position. Muscle tone and peristaltic movements of the renal pelvis and ureters are stimulated by our daily activity and better still by regular vigorous exercise. When immobilization of the body is necessary for protracted periods, drainage in the urinary tract is imperfect and chronic infection with calculus formation is not uncommon.

SUMMARY AND CONCLUSIONS

1. Urinary infections in varying degrees of severity are occasionally found following almost any type of surgical procedure.

2. Severe forms of postoperative infection in the urinary tract are usually the result of postoperative distention.

3. Prompt relief of postoperative urinary retention, accompanied by the administration of a urinary antiseptic, will prevent the severe forms of infection. Catheterization should be carried out early.

4. Prolonged periods of bed rest or immobilization of the body by splints, frames, etc., tend to promote urinary stasis. This stasis may result in infection or stone formation.

REFERENCES

1. CABOT, H. Blood stream infections of the kidney. *Brit. J. Urol.*, 8: 233–256 (Sept.) 1936.
2. CABOT, H. Management of postoperative urinary retention. *Proc. Staff Meet., Mayo Clin.*, 8: 173–175 (March 22) 1933.
3. BRAASCH, W. F. Clinical data concerning chronic pyelonephritis. *J. Urol.*, 37: 22 (Jan.) 1938.
4. HELMHOLZ, H. F. Bactericidal power of urine after use of prontylin by mouth. *Proc. Staff Meet., Mayo Clin.*, 12: 244–245 (April 21) 1937.
5. ROSE, D. K. Importance of bladder function in dealing with infection. *J. Mississippi M. A.*, 34: 33–37 (Feb.) 1937.
6. MELTZER, M. Chronic alkaline encrusted cystitis; vitamin A regime and indwelling catheter. *New York State J. Med.*, 37: 865–868 (May 1) 1937.
7. CABOT, H. Doctrine of prepared soil; a neglected factor in surgical infections. *Canad. M. A. J.*, 11: 610 (Sept.) 1921.
8. BRAASCH, W. F., and COOK, E. N. Infections in the urinary tract. *J. Indiana State M. A.*, 29: 215–217 (May) 1936.

NEW observations support the belief that anterior pituitary extract can cause hypertrophy of the islet tissue of the pancreas.
From—"The Physiology and Pharmacology of the Pituitary Body," vol. 2, by H. B. Van Dyke (University of Chicago Press).

THE ANORECTAL ASPECT OF
VENEREAL LYMPHOGRANULOMA[*][†]

A. W. Martin Marino, M.D., F.A.C.S.

Attending Surgeon, Brooklyn Hospital; Instructor in Surgery, Long Island College of Medicine

BROOKLYN, NEW YORK

AS is well known, up to about five years ago, the general teaching in the United States was that the majority of non-malignant rectal strictures were due to syphilis. The causes of so-called inflammatory rectal strictures, when not attributed to syphilis, were thought to be tuberculous, gonorrheal, or chancroidal infection. Although it is true that occasionally these infections cause rectal stenosis, the experiments, investigations, and reports, here and abroad, in the last twelve years have proved that most of these rectal strictures are manifestations of a protean disease entity, venereal lymphogranuloma.

Historical. Audrey,[1] in 1903, and Puechavy,[1] in 1904, pointed out that the inflammatory rectal strictures which Alfred Fournier[2] described in 1875 and which he named anorectal syphiloma, often accompanied the elephantiasis and progressive ulcerations of the vulva, perineal body, and anal region to which Pierre C. Huguier[3] called attention in 1848 and named esthiomene. In 1913, Durand, Nicolas and Favre[4] fully described the clinical and pathologic features of the inguinal localization of venereal lymphogranuloma. They termed the disease subacute inguinal lymphogranulomatosis and considered it infectious and of venereal origin. In 1925, Frei[5] of Berlin reported a specific intradermal test for this condition. The anorectal aspect of the disease had been considered syphilitic, tuberculous, gonorrheal, chancroidal and even of amebic origin until 1928 when Frei and Koppel[6] discovered that both the inguinal and anorectal aspects of this disease yielded positive reactions to the Frei test. Their observations were confirmed by Jersild[7] in 1930, and by Barthels and Biberstein[8] of Amsterdam in 1931. These findings showed that what primarily had been considered diseased conditions independent of one another were in reality manifestations of a single pathological process of identical etiology.

At the eighth International Congress of Dermatology and Syphilology in Copenhagen, August, 1930, Hellestrom and Wassen[9] announced that they had isolated the virus of this disease. Their investigations were confirmed by Levaditi[10] and his colleagues two months later.

Further studies were carried out and hundreds of reports continued to be published abroad; in 1932 articles began to appear in the American medical literature. The earliest detailed reports in this country were those by De Wolf and Van Cleve,[11] and Sulzberger and Wise.[12] Other articles soon followed, among which were those of Lehmann and Pipkin,[13] Cole,[14] C. F. Martin,[15] Bacon,[16] Bloom,[17] and Grace and Suskind.[18] The enormous amount of literature which has appeared here is proof that the disease is prevalent in the United States, and not confined to the tropics as it was supposed to be. Indeed, it probably occurs with equal frequency in temperate and tropical climates. The real importance of this disease is gradually being recognized in our clinics. The most trying and distressing manifestation is rectal stricture. Chronic ulcerative processes of long stand-

ing most commonly involving the groin, perineal region, vulva and penis, may also appear.

Race. Venereal lymphogranuloma occurs more frequently among colored peoples than in the white race. This does not signify that negroes are inherently more susceptible to the disease than white people. Factors such as sexual habits must be considered. The expression of the disease in both races, however, is markedly different, both as to severity and variations in types of lesions. This probably is due to what Rosser[19] described as fibroplastic diathesis in the negro race, viz., the tendency in colored people to develop thickening, keloidal formations, and pseudo-elephantiasis in infected and injured tissues. In the white race such tendencies are not pronounced.

Nomenclature: Venereal Lymphogranuloma. Numerous terms have been applied to the various aspects of this condition, the most common of which are lymphogranuloma inguinale,[11] lymphopathia venereum,[12] climatic bubo,[20] tropical bubo,[21] non-tuberculous granulomatous lymphadentitis,[22] anorectal syphiloma,[2] esthiomene,[3] and elephantiasis angiomatosa lymphangiectatica lipomatosa fibrosa.[23] These are but a few of the long array of terms. One cannot help but be impressed by the urgent necessity of some general agreement upon the nomenclature. We favor the term venereal lymphogranuloma which was suggested in a recent editorial of the *Journal of the American Medical Association.*[24] If universally adopted, this name would avoid confusion, designate the venereal origin of the disease, give some conception of the nature of the condition, and embrace the inguinal, anorectal, vulval, penile, buccal and submaxillary localizations and aspects of the disease.

Clinical Features of the Anorectal Aspect. Venereal lymphogranuloma is a disease acquired by sexual intercourse and probably caused by an ultramicroscopic filterable virus. The initial lesion, which has an incubation period of from three to seven days, is small and evanescent. It occurs on the penis in males, and usually within the vagina in females. Ten days to a month following exposure, the draining lymph nodes enlarge. From this point, the manifestations of the disease in the two sexes differ.

In the male (Fig. 1), because of the location of the primary lesion, the superficial inguinal nodes become involved and eventually suppurate; secondary perirectal involvement due to the passage of the virus along the collateral lymphatic vessels may occur. Another explanation for rectal involvement in males is sodomy.

In the female (Fig. 2), however, the location of the primary lesion causes the deep pelvic nodes as well as the nodes surrounding the rectum to become involved; inguinal localization is rare. The perirectal involvement produces fibrotic, progressively obliterating, inflammatory rectal strictures, usually accompanied by perianal elephantiasis and often complicated with multiple fistulae. The diseased rectum produces a sanguinopurulent exudate which discharges from the anus. Slight to moderate constitutional symptoms such as chills, fever, and general malaise may be present. The most serious sequela of venereal lymphogranuloma is rectal stricture, the course of which is a very chronic one.

As in syphilis, unusual sex practices cause the initial lesion to appear in extragenital locations. Accidental infections in surgeons[25] as well as in infants[26] have been reported. The lymphatic involvement in these cases naturally is governed by the location of the primary sore.

Diagnosis—The Frei Test. Frei's discovery of his intracutaneous test for venereal lymphogranuloma has made possible our present understanding of the disease. Although, with our present knowledge, the diagnosis of the typical case of rectal stricture due to venereal lymphogranuloma presents little difficulty, there are unusual cases in which the Frei test is invaluable for purposes of differential diagnosis. Un-

doubted cases of syphilitic rectal stricture are rare indeed; the occasional case which is seen requires careful study. The Frei test is of distinct diagnostic value and essential

palpable as well as visible. Of course, control tests are essential. This cutaneous sensitivity lasts for many years, and possibly throughout life.
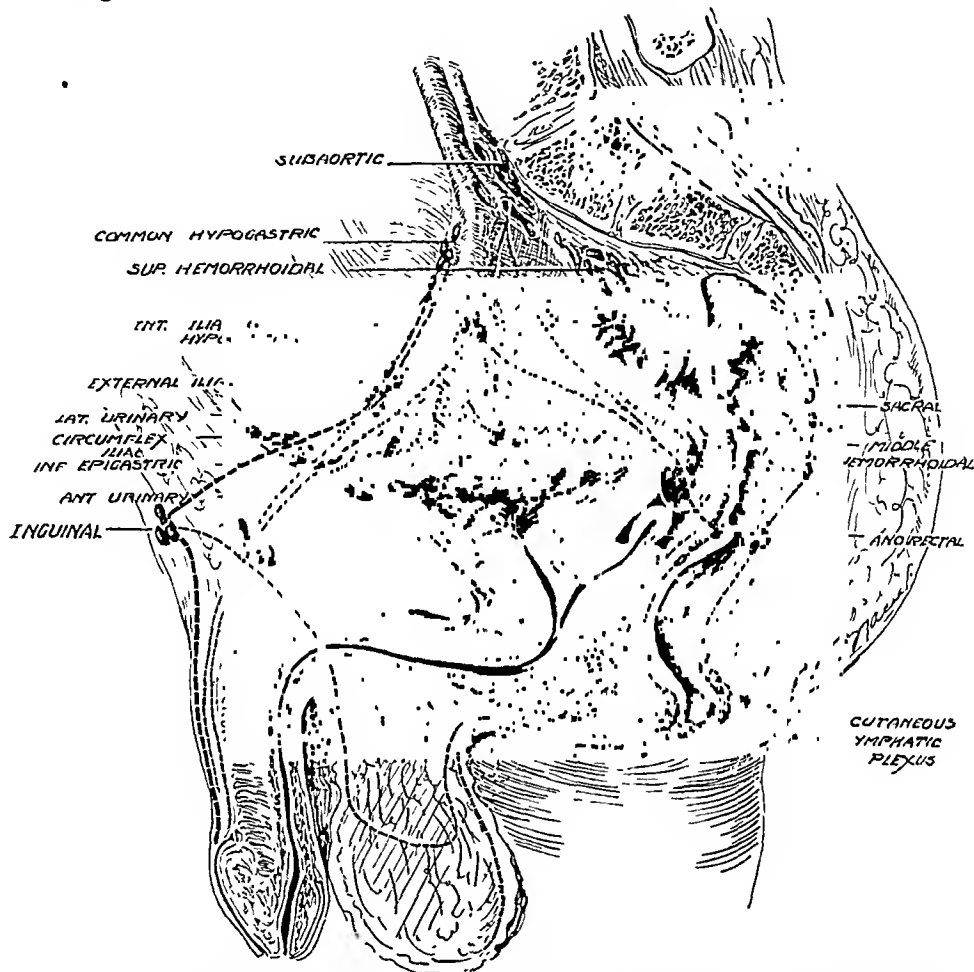

FIG. 1. Cross section of male pelvis showing distribution of lymphatic channels and nodes. After Barthels and Biberstein. (From Lee, H., and Staley, R. W., in *Ann. Surg.*, 100: 490, 1934.)

in these and all other cases in which venereal lymphogranuloma is suspected.

The test consists of the intradermal injection of 0.1 c.c. of antigen prepared from an uncomplicated, fluctuating node in a known case of venereal lymphogranuloma in which there has not been chancroidal, syphilitic, gonorrheal, or, as Frei insisted, tuberculous infection. The reaction should be read after forty-eight to seventy-two hours at which time it reaches its maximum. A positive test is characterized by the production of an infiltrated inflammatory dome-shaped area from 0.5 to 1 cm. in diameter. The reaction is

Preparation of Frei Antigen. According to Frei, the antigen is prepared as follows: Under aseptic precautions, pus from a soft, uncomplicated node (as mentioned above) is aspirated into a syringe. This is diluted and mixed with five to six parts physiologic saline in a sterile tube and immediately put up in 0.5 to 1 c.c. doses in Jena hard-glass ampules. Each ampule is then heated to 60°c. for two hours over a water bath, and the following day at 60°c. for one hour. Before being used, the antigen should be tested for sterility on aerobic and anaerobic media in room and incubator temperature. Tests should then be performed on known

positive cases and normal controls. The potency of the antigen should be tested every three months.

Although the intracutaneous[29,31] and intravenous[32] injections of sterilized Frei antigen offer promise in cases of inguinal
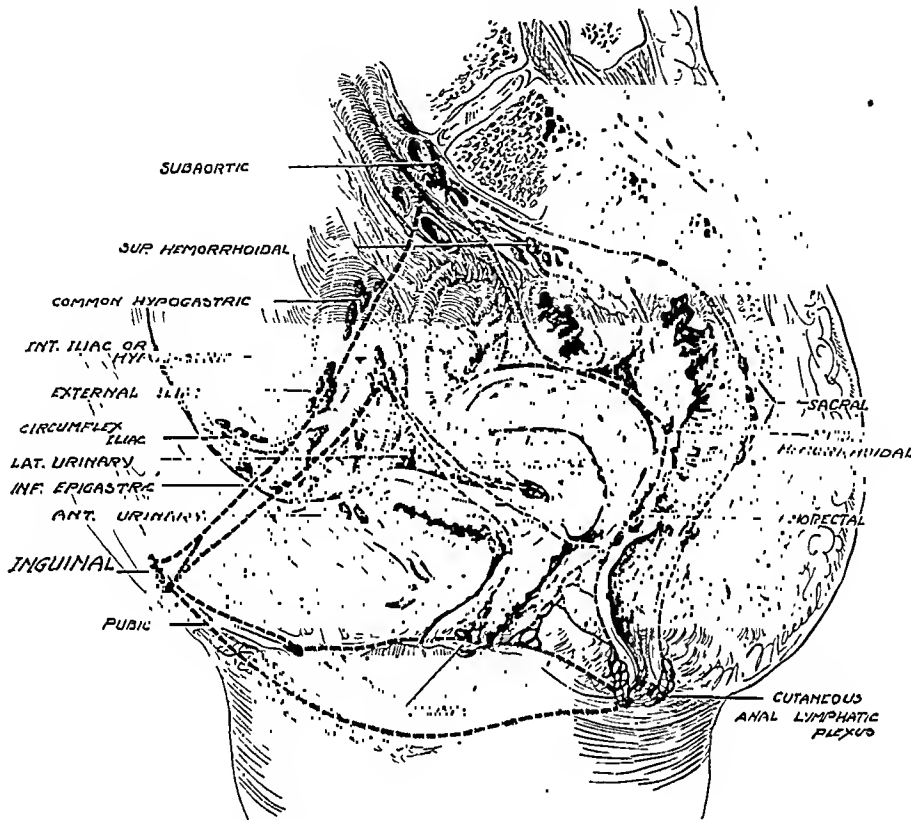

FIG. 2. Cross section of the female pelvis showing distribution of lymphatic channels and nodes. After Barthels and Biberstein. (From Lee, H., and Staley, R. W., in *Ann. Surg.*, 100: 490, 1934.)

Treatment. The remedies suggested for the treatment of this condition are many, but reports of their efficacy are conflicting. No specific has been reported. No routine measure can be recommended. The form and stage of the disease must be considered in selecting therapy. In the absence of suppuration, radiotherapy combined with progressive doses of aqueous solution of iodine associated with sodium thiosulfate is advocated.[27] The favorable results following intravenous injections of tartar emetic[28] and fuadin[29] have not been entirely confirmed. Thomas and McCarthy[30] reported good results in a single case by the use of an autogenous bouillon filtrate applied topically. Hot air and ultraviolet rays as well as injections of patient's own blood and sterile milk have been used.

adenitis, relief from obstruction due to rectal stricture is best obtained by colostomy. For the suppurative lesions, drainage is indicated. Yeomans,[33] in 1933, reported the successful perineal excision of a rectum for stricture which was probably of the venereal lymphogranulomatous variety.

Resection by the abdominoperineal route carries with it a high mortality. Most patients can be made comfortable at least for a time by local surgical measures such as dilatation, posterior proctotomy, internal proctotomy and the excision of elephantiasis and fistulae. We believe, however, that eventually these patients with rectal stricture due to venereal lymphogranuloma must submit to colostomy.

TABLE I

SUMMARY OF FOURTEEN CASES

Case	Race	Sex	Age	Clinical Condition*	Duration		Wass.	G. C.	Frei Test	Treatment
					Sore	Rectal Stricture, Yrs.				
C. J.	C	F	34	Stricture grade 4; esthiomene.	Unknown	3	Neg.	Pos.	Pos.	Colostomy.
A. W.	C	F	46	Stricture grade 4; esthiomene; fistulae.	Unknown	21	Neg.	Neg.	Pos.	Colostomy; local operations; dilatations; tartar emetic intravenously.
V. P.	C	F	41	Stricture grade 2; esthiomene.	Unknown	2	Pos.	Neg.	Pos.	Local operation including internal proctotomy; neoarsphenamine and mercury.
C. C.	C	F	36	Stricture grade 4; esthiomene; fistulae.	Unknown	10	Neg.	Neg.	Pos.	Local operations; refused colostomy.
D. C.	C	F	35	Stricture grade 4; esthiomene.	Unknown	12	Neg.	Neg.	Pos.	Refused colostomy.
M. B.	C	F	31	Stricture grade 4; esthiomene; rectovaginal fistula.	Unknown	4	Neg.	Neg.	Pos.	Local operation; refused colostomy.
J. E.	C	F	38	Stricture grade 2; anorectal fistula	Unknown	3	Pos.	Neg.	Pos.	Local operation; neoarsphenamine and mercury.
C. S.	W	F	49	Stricture grade 4; esthiomene.	Unknown	20	Neg.	Neg.	Pos.	Colostomy; posterior proctotomy; tartar emetic intravenously.
A. G.	W	F	43	Stricture grade 4; anorectal fistulae.	Unknown	18	Neg.	Neg.	Pos.	Colostomy; local operation; tartar emetic intravenously.
M. S.	W	F	30	Stricture grade 2; esthiomene.	10 yrs.	6	Neg.	Neg.	Pos.	Local operation; Frei antigen intracutaneously.
W. B.	W	F	34	Stricture grade 2; multiple abscesses and fistulae.	Unknown	4	Neg.	Neg.	Pos.	Local multiple operations.
K. W.	W	M	36	Stricture grade 2; fistulae.	17 yrs.	2	Neg.	Neg.	Pos.	Local operation.
A. T.	W	M	31	Stricture grade 3; heroin addict 14 yrs.	14 yrs.	4	Pos.	Neg.	Pos.	Posterior and internal proctotomy.
N. T.	C	M	39	Stricture grade 1; penile, scrotal, and anal elephantiasis.	Unknown	7	Neg.	Neg.	Pos.	No surgery. Tartar emetic intravenously.

* Gradation of rectal strictures:
Grade 1: Admitting 1 inch proctoscope with ease.
Grade 2: Admitting $\frac{3}{4}$ inch proctoscope, but not 1 inch proctoscope.
Grade 3: Admitting $\frac{3}{8}$ inch proctoscope with ease, but not $\frac{3}{4}$ inch proctoscope.
Grade 4: Impossible to introduce, or introduction with difficulty of $\frac{3}{8}$ inch proctoscope.

ANALYSIS OF FOURTEEN CASES

We have encountered eighteen cylindrical, inflammatory strictures of the rectum in the last four years. All were subjected to the Frei test. Fourteen of these patients gave strongly positive Frei reactions with different antigens, while repeated tests in the remaining four were negative. Only the fourteen with positive Frei tests will be considered in this summary. Nine of these patients were discovered in the Rectal Clinic of the Brooklyn Hospital, while the remaining five were seen in private practice.

TABLE II

Race	Females	Males	Totals
Colored	7	1	8
White	4	2	6
Totals	11	3	14

Of this group, eleven were females and three were males. The race distribution in the females was seven colored to four white, and in the males one colored to two white. Our finding of such a high incidence of this disease among white people is contrary to the published reports of most writers in the United States. We believe that this seeming discrepancy can be explained by the fact that the percentage of colored applicants to our clinic is relatively low. They are more apt to attend the municipal institutions of our community. We fully realize that no conclusions can be formulated from such a small group of cases as ours, but the findings do emphasize that the disease occurs fairly frequently in the white race.

There were two positive Wassermann reactions among the females, and those occurred in colored women, while there was only one positive Wassermann among the males, and that occurred in a white man. There was no history of chancroid in any of our cases. One of the colored women had a positive vaginal smear for gonorrhea; her rectal smears, however, were negative. No evidence of pulmonary tuberculosis was found in any of our cases, either clinically or on x-ray. Rectal smears proved negative for the Entamoeba histolytica.

It has been stated[34,35] that these patients are from the lower walks of life, but five of our cases definitely did not belong in that category. In fact two of our patients were rather cultured individuals.

Our youngest patient was 30 years of age, and the oldest 49. The average age was about 37. From this it can be deduced that the rectal manifestations in our cases occurred years after the beginning of sexual activity. It is practically impossible to determine with accuracy how soon after the primary infection the rectum becomes involved. These cases are seen only after symptoms such as bleeding, pain, discharge, and obstipation due to partial rectal obstruction occur. Disease in and around the rectum is probably present for years before there are any subjective symptoms.

A definite history of a primary lesion was not elicited in any of our cases. In three cases, however, a history of a small erosion, which we presumed to be due to venereal lymphogranuloma, was obtained. In a male who admitted sodomy, the initial lesion probably occurred in the rectum.

Our patients, according to their histories, harbored these strictures from two to twenty-one years. The average duration was a little over eight years. Generally, as would be expected, the longer the duration of the stricture the more marked and complicated it would be.

Biopsies revealed a remarkable uniformity in the microscopic findings. Sections showed fibrotic tissue containing numerous focal collections of lymphocytes, plasma cells, and other leucocytes, with groups of epithelioid cells in variable numbers. Relatively few foreign body giant cells are present. Different pathologists viewing our sections have called the process infectious granuloma. It was difficult to distinguish the microscopic findings from tuberculosis, syphilis, or other infectious conditions. A knowledge of the clinical findings and a

positive Frei test are supportive in arriving at the histologic diagnosis. The greatest value of biopsy is that malignancy, with which these strictures may be confused clinically, can be excluded.

In the treatment of these cases we have employed the various chemotherapeutic, biologic, and physical measures which have been recommended. We have found them unsatisfactory. Such treatment cannot be expected to alter the effects of fibrosis and cicatrization such as is found in rectal stricture. Surgery is indicated. Colostomy or local surgical measures, or a combination of both, have given our patients symptomatic relief, but the rectal lesions do not show much regression. In cases with imminent obstruction, we have performed colostomies followed by local surgical procedures to establish drainage and reduce the deformity of esthiomene. In cases in which there has not been danger of complete obstruction, we have used such palliative measures as posterior or internal proctotomy in order to postpone the necessity for an artificial anus. Repeated dilatations, in patients with or without colostomy, have not proved of any value. As stated above, we feel that all these cases must eventually submit to colostomy. We have not performed any abdominoperineal resections for these strictures, but it would seem that such operations are feasible.

SUMMARY

Venereal lymphogranuloma, often termed lymphogranuloma inguinale, lymphopathia venereum, etc., is a venereal disease probably caused by a filterable virus. The universal adoption of the term venereal lymphogranuloma would avoid confusion, designate its venereal origin, it would give some conception of the nature of the condition, and embrace the multiple localizations and aspects of the disease.

A history of an initial lesion is difficult to elicit because of the evanescent nature of the primary sore. It occurs on the penis in males and usually within the vagina in females. The manifestations of the disease in the two sexes differ because of the different anatomic locations of the initial lesions. In the male, the superficial inguinal lymph nodes become involved; in the female, however, the deep pelvic nodes as well as the nodes surrounding the rectum become involved. Secondary perirectal involvement may occur in males due to the passage of the virus along collateral lymphatic vessels. In our cases, rectal involvement occurred about four times as frequently in females than in males.

The most serious sequela of the disease is rectal stricture which is the result of perirectal involvement producing obliterating, inflammatory narrowing of the rectal lumen.

This disease probably occurs with equal frequency in temperate and tropical climates. It occurs more frequently in the white race than has been suspected. Just as in syphilis, this disease occurs in people in all walks of life.

The course of the stricture is very chronic; the average duration in our cases was eight years.

Although there is a remarkable uniformity in the microscopic findings, a positive diagnosis cannot be made from biopsy alone.

The Frei test is specific for venereal lymphogranuloma. All rectal strictures require this test.

No specific for the treatment of rectal strictures due to venereal lymphogranuloma has been reported. We believe that these patients must eventually submit to colostomy.

REFERENCES

1. Audrey and Puechavy. Cited by Stannus, H. S. A Sixth Venereal Disease, p. 154. London, 1933. Bailliere, Tindall and Cox.

2. Fournier, A. Cited by Stannus, p. 151.

3. Huguier, P. C. Memoir sur l'esthiomène. Mém. Acad. nat. de méd., 14: 501, 1849.

4. Durand, M., Nicolas, J., and Favre, M. Lymphogranulomatose inguinale subaigue d'origine génitale probable, peut-être vénérienne. Bull. et mém. Soc. méd. d. hôp. de Paris, 35: 274, (Jan. 31) 1913.

5. FREI, W. Eine neue Hautreaktion bei Lympho-granuloma inguinale. *Klin. Wchnschr.*, 4: 2148–2149 (Nov. 5) 1925.

6. FREI, W., and KOPPEL, A. Ulcus chronicum Ele-phantiasticum (Esthiomene) und Soggennantes Syphilome ano-rectal als Folgeerscheinungen der Lymphogranulomatosis inguinalis. *Klin. Wchn-schr.*, 7: 2331 (Dec. 2) 1928.

7. JERSILD, O. Les intradermo-réactions dans le choure mou et dans la lymphogranulomatose inguinale considérées spécialement dans leurs rapports avec l'étiologie du syphilome ano-rectal. *Ann. dermat. et syph.*, 1: 577–608 (June) 1930.

8. BARTHELS, C., and BIBERSTEIN, H. Zur Ätiologie der "entzuendlichen" Rectumstricturen. (Lym-phogranulomatosis inguinalis als Grundkrank-heit.) *Beitr. Z. klin. Chir.*, 152: 161, 1931.

9. HELLESTROM, S., and WASSEN, E. Meningo-enzephalitische veranderungen bei Affen nach intra-cerebraler Impfung mit Lymphogranuloma inguinale. *Verb. 8th Internat. Konbr. Dermat. u. Syph.*, Kopenhagen, August 5–9, 1930.

10. LEVADITI, C., et al. Cited by Stannus, H. S.[1]

11. DE WOLF, H. F., and VAN CLEVE, J. V. Lympho-granuloma inguinale. *J. A. M. A.*, 99: 1065 (Sept. 24) 1932.

12. SULZBERGER, M. B., and WISE, F. Lymphopathia venereum. *J. A. M. A.*, 99: 1407 (Oct. 22) 1932.

13. LEHMANN, C. F., and PIPKIN, J. L. Lymphopathia venerea. *Texas State J. Med.*, 29: 192–199 (July) 1933.

14. COLE, H. N.: Lymphogranuloma inguinale, the fourth venereal disease. *J. A. M. A.*, 101: 1069 (Sept. 30) 1933.

15. MARTIN, C. F. Stricture of the rectum. Some of its problems. *J. A. M. A.*, 101: 1550 (Nov. 11) 1933.

16. BACON, H. E. The specificity of the Frei test in lymphopathia venerea. *Tr. Am. Proc. Soc.*, p. 97, 1935.

17. BLOOM, D. Strictures of the rectum due to lym-phogranuloma inguinale. *Surg., Gynec. & Obst.*, 58: 827–840 (May) 1934.

18. GRACE, A. W., and SUSKIND, F. H. Lympho-granuloma inguinale. *Arch. Dermat. & Syph.*, 34: 65 (July) 1936.

19. ROSSER, C. Rectal pathology in the negro. *J. A. M. A.*, 84: 93 (Jan. 10) 1925.

20. RUBER, J. Cited by Bacon and Meharg.[29]

21. LEJARS, F. Cited by Bacon and Meharg.[29]

22. HANSMANN, G. Non-tuberculous granulomatous lymphadenitis. *Surg., Gynec. & Obst.*, 39: 422, 1924.

23. RAVOLGLI, A. Cited by Stannus, H. S.[1]

24. Editorial. Venereal lymphogranuloma. *J. A. M. A.*, 109: 876 (Sept. 11) 1937.

25. HELLESTROM, S. Cited by Thompson.[27]

26. LUJAN and ROTTER. WALLIS, F. C. Cited by Stan-nus.[1]

27. THOMPSON, R. M. Lymphopathia venereum. *J. A. M. A.*, 106: 1869 (May 30) 1936.

28. MARTIN, C. F., BACON, H. E., and NESSELROD, J. Cited by Bacon and Meharg.[29]

29. BACON, H. E., and MEHARG, J. G. Lymphopathia venerea. *Med. World*, 53: 711 (Nov.) 1935.

30. THOMAS, W. A., and MCCARTHY, E. R. Treatment of lymphogranuloma inguinale with bouillon filtrate. *J. A. M. A.*, 102: 766 (March 10) 1934.

31. PREHN, D. T. Lymphogranuloma venercum and associated diseases. *Arch. Dermat. & Syph.*, 35: 231 (Feb.) 1937.

32. GAY-PRIETO, J. Cited by Prehn.[31]

33. YEOMANS, F. C. Stricture of the rectum; result of twenty-three years after perineal excision. *Tr. Am. Proc. Soc.*, p. 70, 1933.

34. ALLEY, R. C. Rectal stricture. Relation to lympho-pathia venerea. *Tr. Am. Proc. Soc.*, p. 150, 1934.

35. LEE, H., and STALEY, R. W. Inflammatory stric-tures of the rectum and their relation to lympho-granuloma inguinale. *Ann. Surg.*, 100: 490, 1934.

PERFORATION OF THE GALL-BLADDER

WADE W. STONE, M.D.   AND   FRED M. DOUGLASS, M.D.

Attending Surgeon, St. Vincent's and Toledo Hospitals   Director of Surgery, St. Vincent's Hospital

TOLEDO, OHIO

PERFORATION of the gall-bladder is too frequently considered one of the rare complications in gall-bladder surgery, probably because perforation is usually reported in conjunction with cholecystitis in its chronic state. When one considers the acute gall-bladder and the frequency with which it ruptures, one cannot regard perforation as a rarity. The commonly quoted statistics of perforation show that it occurs in from 1 to 2 per cent of chronic cholecystitis. In 6,800 cases reported by five authors (quoted by Eliason & McLaughlin[1]) there were ninety-six perforations (0.9 to 2.5 per cent). Recently Sanders[2] reported an incidence of 5.2 per cent in a series of 886 cases.

We are reporting a group of seventeen cases in which rupture occurred, in a series of 775 gall-bladder operations done from June, 1925 to January, 1934, the percentage in this series being 2. However, 170 of the total number were acute cases, which brings the incidence to 10 per cent. This is not so high as reported by Heuer,[3] whose figure of 26 per cent is greater than that occurring in Zinninger's[4] series (20.5 per cent) and in Smith's[5] series (22.4 per cent). Judd and Phillips[6] report an incidence of gangrene and perforation in acute cholecystitis of 13.4 per cent, and qualify their findings, stating that because of their location they did not see so many acute cases as surgeons in larger communities. Our incidence of acute gall-bladders presents a figure of 22 per cent, which is more in keeping with that of perforation as reported by the previously mentioned surgeons; but our percentage of perforations we think is still of sufficient size as to make one give much thought to the acute gall-bladder. Seven of the cases occurred in males, ten were in females. The average age was 52; the youngest patient was 23 years of age and the oldest 75. The mean age for the entire group of acute cases without perforation was 46.

Signs and Symptoms. Pain was present in all cases. Practically all had a history of preëxisting chronic cholecystitis. The onset of the present illness occurred usually two or more days before the patient was seen by us. Jaundice was present in but six cases. Twelve had vomited. Chills were noted in but two cases. The average temperature on admittance was 99°F., the highest being 102 degrees, and there were eight cases with normal temperature; temperatures were the same for both the acute and perforated gall-bladders. There was localized tenderness over the gall-bladder in all but two cases, and in these there was generalized tenderness.

The average white count was 14,000, the lowest being 4,500 and the highest 25,000. The mean white count for the total series of acute cases was 11,000, as compared with 14,000 for those with perforated gall-bladders.

Thirteen patients had stones and four gall-bladders perforated in the absence of stones; in only one case were there stones in the common duct. Perforation of the gall-bladder into the free abdominal cavity of a patient in the third trimester of pregnancy has been reported by one of us.[7] Diabetes complicated but one case in this series.

Preparation of Patient. Many times the patient was in such poor general condition that operation was withheld for a period of eight to ten hours, during which time the patient was given large amounts of glucose, saline, and in some cases blood transfusion. It is our belief that in acute gall-bladders,

the sooner surgery is done the better the end result, and this certainly is even more true when perforation of the gall-bladder has occurred. Many times the diagnosis of acute gall-bladder disease is difficult to make, but whenever a patient presents himself with a history of chronic gall-bladder disease, with severe pain in the right upper quadrant, low fever, elevated white count, and with a tender mass or rigidity in the right upper quadrant, surgery should be done promptly. We find it very difficult to diagnose perforation in advance of operation, and consider it even more difficult to predict which gall-bladder will go on to perforation and which will become quiescent, for in our small series we have perforations of the gall-bladder with normal temperatures and normal white counts, and therefore feel the best time to operate on the acute gall-bladder is before it has become either gangrenous or perforated.

Type of Operation.   Whether removal of the gall-bladder or simple drainage is to be done depends first, on the patient's general condition and second, on the pathology present. In the aged individual whose general condition is poor, cholecystostomy under local anaesthesia with drainage of the abdomen, is sometimes all the surgery permissible. Occasionally when gangrene is so extensive as to make cholecystectomy extremely difficult, once again the easier of the two operations is done. In this series, eleven gall-bladders were removed and six were drained. Of this latter group, two gall-bladders have since been removed.

Postoperative Condition of Patient.   The convalescence, while quite stormy the first two or three days, was frequently not so severe as anticipated. The highest mean temperature postoperatively was 101 degrees. We feel that the patient who has had a suppurative and perforated gall-bladder removed will have a smoother convalescence than one whose gall-bladder was merely drained. The high fever described in the past as liver shock was not seen in any

of these cases, which may be explained either by the administration of large amounts of glucose to prevent glycogen depletion of the liver, or by taking particular care that the right hepatic artery is not ligated. Eleven of the seventeen patients had distention which might be described as moderate. There was drainage from the abdominal wound in the average case for twenty-two days, all wounds being considered infected.

Complications.   In five cases there were definite chest complications, only two of which were true pneumonia. Diathermy was given with apparent success. Since this series, postoperative inflation of the chest is done routinely and we believe has reduced our incidence of complications of this type. Two subphrenic abscesses occurred postoperatively and were drained; one of these patients died fifty-five days following the original operation and was our only mortality. The diabetic, whose blood sugar at one time was 222 mg., made an uneventful convalescence. The draining infected wounds prolonged the patients' stay in the institution and were the ultimate cause of five incisional herniae. The case of perforated gall-bladder complicating the last stage of pregnancy went on to a very satisfactory termination for both the mother and child.

Site of Perforation.   In our experience, the majority of gall-bladders perforate at the so-called "gooseneck," but perforation may readily occur at the fundus, also. The absence of stones does not preclude the possibility of perforation, as in four cases this occurred when no stones were present. Whether or not there is free perforation into the abdominal cavity has much to do with the mortality in perforation of the gall-bladder, according to the literature as reported by Mitchell[8] and Alexander.[9] The figure fifty is the most frequently quoted mortality incidence when there is contamination of the abdominal cavity by bile from a perforated gall-bladder. In our series, perforation into the free abdominal

cavity occurred in six cases without a death. We like to believe that the early institution of operation was a big factor in this achievement. Eleven were walled off by some adjacent organ or structure so as to produce extracholecystic abscess. Two of these eventually went on to subphrenic abscess formation with one death. In none of these cases was there gross hemorrhage into the abdomen.

Mortality. Of the seventeen cases of perforated gall-bladder, there was one death, which makes a percentage of 5.8.

SUMMARY

1. Seventeen cases of perforation of the gall-bladder are presented, with but one mortality.

2. Four cases of perforation of the gall-bladder in the absence of stones were encountered, this being distinctly in disagreement with the early teaching concerning perforation of the gall-bladder.

3. Six cases of acute free perforation of the gall-bladder without a death are reported.

CONCLUSION

A plea is made for earlier operation in acute gall-bladder disease, so that the organ can be removed rather than drained; and complications such as extracholecystic abscess, liver abscess, and fistulae between the gall-bladder and neighboring viscera will not be met, as these are the complications which do much to increase morbidity and mortality in perforation of the gall-bladder.

REFERENCES

1. Eliason, E. L., and McLaughlin, C. W. Perforation of the gall-bladder. *Ann. Surg.*, 99: 914, 1934.
2. Sander, R. L. Perforation of the gall-bladder. *Surgery*, 1: 949, 1937.
3. Heuer, G. J. Surgical aspects of acute cholecystitis. *Ann. Surg.*, 105: 758, 1937.
4. Zinninger, M. Treatment of acute cholecystitis. *Ann. Surg.*, 96: 406, 1931.
5. Smith, M. K. Treatment of acute cholecystitis. *Ann. Surg.*, 98: 766, 1933.
6. Judd and Phillips. Acute cholecystic disease. *Ann. Surg.*, 98: 771, 1933.
7. Stone, W. W. Perforation of the gall-bladder occurring in the late stage of pregnancy. *J. A. M. A.*, 109: 1903, 1937.
8. Mitchell, E. *Ann. Surg.*, 88: 200, 1928.
9. Alexander, E. G. *Ann. Surg.*, 86: 765, 1927.
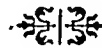
THE MORTALITY AND MORBIDITY OF ACUTE APPENDICITIS

A STUDY OF 150 CASES

Arthur B. Raffl, M.D., F.A.C.S.

SYRACUSE, NEW YORK

IN the four or five decades that have passed since the treatment of acute appendicitis was placed on a surgical basis, many statistics have been given in the literature on the operative mortality of this disease. Mortality rates varying from 3 to 10 per cent are reported[1] from studies of large series. Most of these studies were made on the results obtained on combined ward and private services and probably do not present a true picture of the surgical treatment of appendicitis at its best. More recently a report by Herrick[2] and another by Bliss and Heaton[1] show much lower mortality figures. In the latter series, a study of appendicitis in the army service, most of the cases were seen and operated on early and for that reason perhaps the excellent results were obtained. However, it is a noteworthy fact that in these and other[3,4,5] series with low mortality the great majority of the patients were operated on through a McBurney incision or a modification of it. Reid[6] says that the operative mortality of appendicitis has dropped over 50 per cent in the Cincinnati General Hospital since the McBurney replaced the right rectus incision. Furthermore, these observers follow a certain régime with which it is difficult to disagree.

Every surgeon must have a sort of surgical creed in dealing with appendicitis. It is only through following the tenets of his faith that he may hope to achieve the best results.

This study is based on a review of 150 consecutive private cases treated by operation in one hospital. Every case presented a picture of acute inflammation to the pathologist.

Before reviewing the statistics, one must take into account some of the things which may have a bearing on the results. There are, first of all, factors over which the surgeon has no control. There can be no question that the time elapsing between onset and operation is very important. The taking of cathartics and, possibly, of food aggravates the condition. The age of the individual and his resistance to infection play an important rôle. Finally, the type of infecting organism certainly has to be considered.

Then there is the old controversy of the delayed versus the immediate operation. Without entering into a discussion of the merits of one or the other, we take the side of those who operate as soon as possible or just as soon as the very sick patient is in condition. Nothing except impending collapse should delay removal of the offender. If the removal is done properly there will be very little spread of the peritonitis from the operative procedure. Those patients who come into the hospital with peritonitis, dehydration and signs of circulatory failure should be treated with intravenous solution of 5 per cent glucose in saline plus blood transfusion, until they show improvement.

In our series, the anesthetic used was a combination of regional block with 1 per cent novocaine and adrenalin and inhalation of nitrous oxide, ethylene, or, more recently, cyclopropane.

Except when the diagnosis was in some doubt or when a mass was located too far medially, a transverse muscle-splitting incision was employed. The site of this incision varied a little, depending on the location of a mass but usually it was placed

at the level of the anterior superior iliac spine. Every effort was made to enter an abscess without opening into the general peritoneal cavity.

All fluid was cultured. As soon as the peritoneal cavity was entered, fluid, if present, was aspirated. The cecum was located and the longitudinal bands followed to the base of the appendix. The tip was not delivered unless it came up easily. Quite frequently the appendix was removed in retrograde fashion by ligating its base and severing with a carbolized knife, after clamping securely to avoid spilling its contents. The mesoappendix then was clamped and cut a bit at a time, thus freeing the appendix with a minimum of manipulation. The utmost gentleness was employed to deliver the organ intact. A small light-equipped ribbon retractor sometimes was very useful, but generally retractors and gauze packs were avoided. When the omentum was attached, it was ligated, cut away from the main portion, and left adherent in one piece with the appendix. The stump was inverted with an N-suture of fine silk if there was no inflammatory thickening of the cecum and no abscess or peritonitis. The appendix was removed in every case except one.

All patients with abscess or peritonitis were drained. Where there was enough peritoneal reaction to produce seropurulent fluid with no perforation, closure was done without drainage. The position of the drains was determined to some extent by the findings, but usually one was placed lateral to the cecum, the other toward the pelvis. Large cigarette type drains were used.

In the ordinary case the peritoneum was closed with a plain No. o catgut continuous suture. Interrupted chromic catgut No. o sutures approximated muscle and fascia and the skin was closed with Bartlett clips. When drainage was employed, one or two dermal stay sutures sufficed to hold the entire abdominal wall down to the peritoneum, the drains coming out of the wound at its lateral aspect. Thus the tissues of the abdominal wall were left free to fight infection and to drain without as much danger of sloughing. Because of the nature of the incision, with its tendency to close rather than open with straining, no fear of eventration was felt.

In the uncomplicated case without drainage, the patient was given water by mouth as soon as nausea had ceased. He received fluid food after twenty-four hours and a soft diet soon afterward. If there was any question of proper ingestion, the giving of food was delayed. Until the patient was able to take fluids by mouth he was given 3,000 c.c. of 5 per cent glucose in saline intravenously every twenty-four hours. For sedation morphine in ⅙ or ¼ gr. doses was used. On the third day following the day of operation he received an enema if he had not defecated naturally. During the first few days he was made to take a few deep breaths every hour. The Bartlett clips were removed on the fourth day, he was up in chair on the fifth and usually he left the hospital on the seventh or eighth day.

Of much greater importance is the postoperative care of the patient in whom the infection has spread to the peritoneal cavity. It is in this type of case that the mortality and morbidity definitely may be lowered by proper treatment. These patients received absolutely nothing by mouth. If they were distended or were vomiting, the Wangensteen type of suction drainage was instituted. Fluid by mouth was not started until improvement was certain. It is often disastrous to begin the oral administration of fluid and food too early. Morphine was administered hypodermically in ⅙ or ¼ gr. doses often enough to keep the respirations to sixteen per minute. Every twenty-four hours the adult patient received 3,500 to 4,000 c.c. of fluid intravenously. This fluid was 5 per cent glucose either in saline or in distilled water. More saline was used if there was a large loss of upper intestinal content, less if there was any evidence of edema. The urine output was followed as a good guide to amount of intake. It was kept as near as

possible to 1,500 c.c. daily in adults. The patient was kept in a semi-sitting position. Large, hot, moist dressings were applied to

(Chart II.) Whether the very young and the aged are more susceptible to rupture or whether diagnosis is more difficult in these
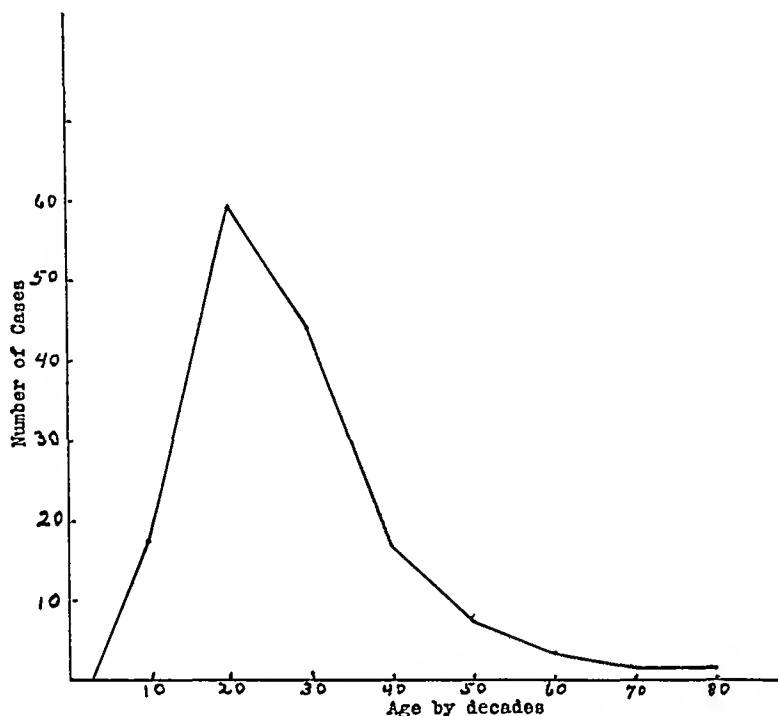

CHART I. Occurrence of appendicitis as related to age.

the abdomen at two-hour intervals. These extended from xiphoid to symphysis and covered the entire abdomen. The wound was uncovered entirely with each change. A rectal tube was inserted if there was distention but enemata never were given during the acute stage. Deep breathing at frequent intervals was encouraged and the legs were massaged and exercised twice daily. If the patient was very ill or if his illness was prolonged, he received whole blood transfusions. During the entire convalescence a constant vigilance was kept for complications. Residual abscesses were drained by the most direct route. Most of these were pelvic and were drained through the rectum.

There was a sharp rise in the number of cases in groups from 10 to 30 years of age, with a preponderance in the second decade. (Chart I.) These findings are in accord with those of others. In contrast, there was a sharp fall in the percentage of cases of ruptured appendices in this age group.

cases has to be considered. Probably both factors enter the picture.

The cases were about evenly divided as to sex, seventy-three being male and

TABLE I

Sex { 73 male / 77 female

Average Hours Before Operation
Non-ruptured..... 24 (earliest 3 hrs.)
Ruptured......... 104 (earliest 13 hrs.)

History of Previous Attacks
Non-ruptured......... 58—47 per cent
Ruptured............ 11—40 per cent

Average Hospitalization
Non-ruptured................ 8 days
Ruptured.................... 19 days

Right Paramesial Incision
11 cases
Ruptured cases { 17 male / 10 female

History of Catharsis
Non-ruptured.... 13 cases—10 per cent
Ruptured........ 12 cases—44 per cent

Average Duration of Operation
Non-ruptured—18 min. { Shortest—8 min. / Longest—40 min.
Ruptured—26½ min. { Shortest—10 min. / Longest—45 min.

Palpable Mass
16 cases

seventy-seven female. (Table I.) Seventeen of the ruptured appendices were in males and ten in females. In the non-ruptured cases an average of twenty-four hours had elapsed before operation. The earliest was operated three hours after the onset. An average of 104 hours from onset to operation had elapsed in the cases with rupture. One patient showed perforation though only thirteen hours had elapsed and no cathartic had been given.

There was a history of catharsis in twenty-five cases. Among the ruptured appendices, twelve cases, or 44 per cent, followed a cathartic.

Previous attacks had occurred in fifty-eight or 47 per cent of the unruptured cases and in eleven or 40 per cent of those that had perforated.

One of the many advantages of the McBurney type of incision is the ease with which the abdomen may be opened and closed. The operating time, therefore, is cut down, a factor of no little importance in acute cases. The average time for the non-ruptured cases was eighteen minutes. The average of twenty-six and one-half minutes for the ruptured cases shows quite clearly how the operative procedure is affected by the pathology.

Another very definite advantage of the muscle-splitting incision is its ability to stand stress and strain rather early as compared to other types. As a result, patients may be walking on the sixth postoperative day and frequently may leave the hospital on the seventh day. The average hospitalization period for the non-ruptured cases was eight and one-half days. The morbidity of rupture is demonstrated by the prolonged hospitalization. The average in these cases was nineteen days.

There was a palpable mass in sixteen of the cases with rupture. Complete localization was not present, but rather there was some attempt at localization.

A right paramesial incision was employed in eleven cases, either because of some doubt as to diagnosis as mentioned before,

or because it offered a more direct approach to a medially placed mass.

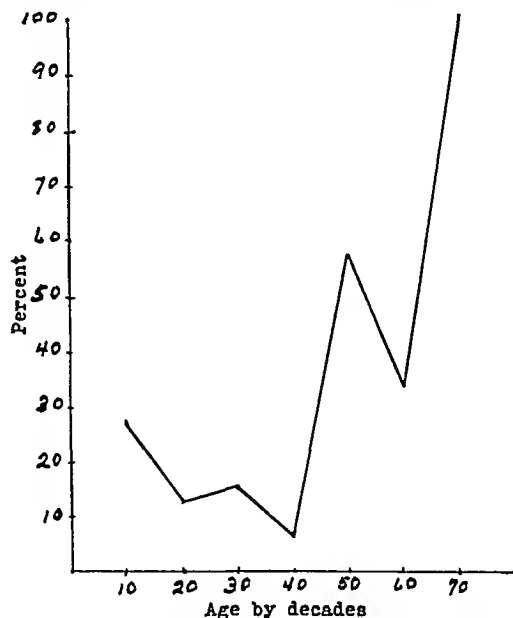Cultures were made of all fluid. There

CHART II. Percentage of ruptured appendices as related to age.

were fourteen patients with fluid but without definite perforation. These were not drained. In one case hemolytic streptococcus was cultured, in one Staphylococcus aureus, and two gave a pure growth of Bacillus coli communis. All of these recovered without complication except for a wound infection with colon bacillus.

Twenty-five of the ruptured cases gave positive cultures. As far as could be determined from observation of the postoperative course, the type of organism did not seem to affect it except in one patient infected with Bacillus mucosus capsulatus. This very interesting recovery followed six months of hospitalization, during which time three residual abdominal abscesses and a right subphrenic abscess were drained. Each abscess gave a pure growth of Friedlaender's bacillus. At the operation for obstruction which occurred six months after the appendectomy and drainage, a small unsuspected pelvic abscess was found and it, too, gave a pure growth of this organism.

As a general rule, the white blood cell count followed the pathology fairly well,

but in fifteen cases, or 10 per cent of the series, the count was below 10,000. However, a differential count showed more than 80 per cent polymorphonuclear leucocytes in eight of these. Three of these cases showed considerable gangrene of the appendix. In every case with rupture the leucocytes and the polymorphonuclear percentage were increased.

The chart of complications shows how seriously rupture of the appendix affects the outcome. (Table II.) There were only

TABLE II
COMPLICATIONS
Non-ruptured Cases

Wound infection (colon bacillus)	2
Pregnancy . (4 and 7 months)	2
Bronchitis.	1
Pneumonia .. (Group IV)	1
Acute otitis media.	1
Infarct of lung .. .	1
Postoperative complications . '	6—4 8 per cent
Mortality of series of 150 cases .	0 66 per cent
Mortality of 27 ruptured cases ..	3 7 per cent

Ruptured Cases

Residual abscess.. ...	8
Fecal fistula . .	3
Residual abscesses Subdiaphragmatic abscess⎫ Femoral phlebitis ⎬ . 1 Intestinal obstruction ⎭	
Postoperative hernia. . .... (right rectus incision)	1
Peritonitis 8 days (died)	. 1
Postoperative complications. (including one death)	14, or 52 per cent

six complications among the unruptured cases. Not even the postoperative pneumonia was serious. On the other hand, all the patients with residual abscesses were quite ill. The patient with multiple complications, mentioned previously as infected with Friedlaender's bacillus, was near death several times during his six month illness. Over half of these cases developed one or more complications before they recovered.

One death occurred, in a woman aged 51 who had received chiropractic treatment for a week after the onset of her pain. She had a mass the size of a football in her right abdomen. She was obese and so ill that appendectomy was not attempted. Her abscess was drained and after a period during which she seemed to be recovering, she died of a pulmonary embolus.

The mortality of appendicitis as indicated by this series and others would seem to be the mortality of rupture. So long as the inflammatory process remains confined to the appendix, operative removal is safe and sure, but once the infection spreads to the peritoneum an unfavorable outcome is quite possible.

SUMMARY

A series of 150 consecutive private cases showing the pathology of acute appendicitis is studied for morbidity and mortality.

The method of operation and treatment is given.

Age and sex incidence and other miscellaneous data are charted.

The series shows morbidity in six unruptured and twelve ruptured cases, or 13.3 per cent for the entire series.

The mortality rate is 3.7 per cent for ruptured appendicitis, 0.66 per cent for the series.

REFERENCES

1. BLISS, R. W., and HEATON, L. D. Appendicitis in army service. *Ann. Surg.*, 107: 242–247 (Feb.) 1938.
2. HERRICK, F. C. Acute appendicitis with peritonitis; treatment and mortality. *Surg., Gynec. & Obst.*, 65: 68–72 (July) 1937.
3. WOLFSON, W. L., and MORSE, L. J. Acute appendicitis of the severe type; a review of one hundred consecutive cases with special reference to operative technique. *Am. J. Surg.*, 40: 100–105, 1926.
4. LEONARD, E. D., and DERON, S. Mortality factors in acute appendicitis. *New England J. Med.*, 214: 52 (Jan. 9) 1936.
5. PATTISON, A. C. Mortality of acute appendicitis. *Ann. Surg.*, 103: 362–374, 1936.
6. REID, M. R. Some remarks on the operative procedure for appendicitis. *Surg., Gynec. & Obst.*, 59: 529–532, 1934.

PRINCIPLES OF SURGICAL TECHNIQUE*

WITH PARTICULAR REFERENCE TO THE USE OF SILK

CHARLES H. LUPTON, M.D.

Senior Surgical Staff, Norfolk General Hospital

NORFOLK, VIRGINIA

THE principles of surgical technique here discussed are based to a great extent upon observations and work done at Peter Bent Bringham Hospital, the Harvard Medical School, and in private practice.

Dr. Elliott C. Cutler tells the following story about Dr. Harvey Cushing: When Dr. Cushing had completed his training in surgery at the Massachusetts General Hospital, he received an appointment as assistant to Dr. Halsted at Johns Hopkins. From his training at Massachusetts General, he had come to believe that speed was an important factor, and regarded with considerable pride the fact that he had assisted Dr. J. C. Warren to perform a radical amputation of the breast in some twenty-two minutes. When he went to Johns Hopkins, Dr. Halsted requested him to observe their methods for a few days before beginning to assist. The first morning Dr. Cushing observed an operation, Dr. Halsted was doing an amputation of the breast. When nearly two hours had passed and Dr. Halsted had not completed the operation, Cushing had become rather anxious about the patient's condition. Finally after nearly two and a half hours the operation was completed, and by that time Cushing's anxiety had about reached the limit. Late that afternoon when Dr. Halsted was making rounds he went in to see the patient and found Dr. Cushing there with saline, strychnine, etc., all ready. It was then that Dr. Cushing explained his anxiety concerning the patient and stated he considered her badly shocked. Dr. Halsted told him if she was shocked it could be easily determined by taking her blood pressure. They took the blood pressure and to the amazement of Dr. Cushing it was normal. Then they took up one by one the questions of giving her strychnine and other remedies Dr. Cushing had in the room, and after their discussion, Dr. Cushing was unable to give good reasons why the patient would be benefited by any of them. To Dr. Cushing's surprise and amazement the patient made an uneventful recovery.

Dr. Cushing often tells this story on himself to emphasize that hurry is not a good surgical principle. To be able to operate quickly and at the same time to observe certain well defined principles of surgery such as gentleness and careful hemostasis is fine, but no surgeon should attempt to hurry through an operation in a patient who is a good risk. He cannot hurry and at the same time observe the primary principles of gentleness and careful hemostasis.

There has been voluminous literature during the past two decades or more on the importance of preoperative preparation and postoperative care. I do not desire in any way to detract from the importance of all that has been written; in fact, I think it would be well to reëmphasize and concur in the importance of careful pre- and postoperative measures. But this point I do want to make: if there has been proper preoperative investigation and preparation, and careful technique—including gentleness and hemostasis during operation—then there will be fewer postoperative complications, and the postoperative care will not play so important a part.

I do not expect all surgeons to agree with some of the principles I follow, particularly in the use of silk. I probably would have disagreed with these same statements one year ago, particularly in reference to speed and the use of silk. I had always attempted to place considerable importance on speed, or perhaps what would be nearer correct would be to say speed to the extent of hurrying, and while I had seen some silk used, I have never been convinced of the logic of its use.

It is not my intention to discuss the technique of different surgical procedures. Dr. Cutler says: "If you are gentle, if you do not infect your patient, if you are careful about hemostasis, and have proper anesthesia, all other things being satisfactory in a good risk patient, you can operate on the patient as long as it is reasonably necessary." After observing his technique and seeing his patients after operation, I am inclined to believe what he says. Speed must never be obtained at the sacrifice of gentleness and careful hemostasis. The surprising thing to me is that Dr. Cutler's patients do not appear to be sick after surgical intervention as my patients used to be, and as those of other physicians frequently are. It is unusual to hear his patients complain to any particular extent; they look well, usually smiling, and the record in reference to temperature, pulse, respiration, etc., seems to bear that out. They do not seem to have the complications and distention I have been used to seeing. The temperature seldom goes above 100.5 F. at any time during their postoperative stay in the hospital. The postoperative appearance of patients, who in the vast majority of cases did not appear really sick, and whose charts indicated their unusually mild postoperative courses (much better than I had been accustomed to see), was the thing that convinced me of the logic of his teachings.

What are the factors in producing such satisfactory postoperative courses?

1. Careful hemostasis.
2. Gentleness.
3. The use of silk.

Of course there are other factors such as asepsis, proper anesthesia, etc. But these are observed by many surgeons who nevertheless are not able to obtain the mild postoperative course that is seen where gentleness and careful hemostasis are stressed, and where silk is used. Gentleness and hemostasis employed in the use of catgut are far different from the same connections when silk is used. The full significance of this is not appreciated until silk is actually employed. With silk a gentleness far beyond what one is accustomed to employ is necessary, because of the delicate nature of the fine silk used. As this gentleness improves, one naturally develops greater consciousness for better hemostasis. Gentleness and careful hemostasis go hand in hand with the use of silk.

I saw a bilateral herniorraphy performed, silk being used on one side and catgut on the other, with silk used in the skin on both sides. After three days the dressings were removed and those in the class who were not present when the operations were performed were asked to examine the wounds to see if they could determine on which side catgut had been used. All five men were able to state definitely on which side each had been used. On the catgut side the width of the area of induration and tenderness along the incision was about twice as great as on the silk side.

Dr. Shambaugh has reported on postoperative wound complications in a controlled series of 2,360 inguinal hernorraphies performed from 1914 to 1934, comparing silk and catgut. When catgut was employed, No. 1 or 2 chromic was used in the fascia, and No. 00 plain for the hemostatic ties. When silk was employed No. 4 black twisted silk was usually used, although occasionally No. 9 black twisted silk was used in the fascia.

In the 2,360 operations there were 108 infections, an incidence of 4.57 per cent. There were sixty-one minor wound complications (hematomas or serum), 2.59 per

cent. There were forty-seven suppurative infections, an incidence of 1.99 per cent.

During the past fourteen years there has been a more extensive use of silk and at the same time an almost continuous decrease in the number of infections, so that out of a total of 511 operations during the past five years there were only seven infections (five suppurative infections and two minor wound complications) an incidence of 1.27 per cent. Everything else being equal, it was found that wound infections occurred 30 per cent more frequently in patients 50 years and older than in patients under 50 years of age. (Table II.) The use of local anesthesia was not found to be a factor in the development of wound infection (Table III), but the incidence of wound infection in the catgut series was twice that of the silk series. In minor wound complications the superiority of silk was still greater, as these minor infections occurred eight times more

TABLE I

INCIDENCE OF FAULTY WOUND HEALING IN CLEAN INGUINAL HERNIORRHAPHIES*

Author	Number of Cases	Wound Infections		Total Wound Complications		Method of Skin Preparation
		No	Per Cent	No	Per Cent	
Coley	500	64	12 8	..	..	Iodine
Beckman	308	17	5 5			Not stated
Meleney	502	21	4 1	50	9 9	Ether, soap, iodine
Morian	1407	40	2 8	73	5 2	Not stated
Eliason	749	13	1 7	78	10 4	Soap and mercurochrome
P B B H series	2360	47	1 9	109	4 5	Alcohol and bichloride

TABLE II

EFFECT OF AGE UPON THE INCIDENCE OF WOUND INFECTIONS*

Age Group	Number of Operations	Suppurative Infections	
		No.	Per Cent
Under 50 years.	1730	31	1 79
50 years and over	630	15	2 38

TABLE III

EFFECT OF LOCAL ANESTHESIA UPON THE INCIDENCE OF WOUND INFECTIONS*

Anesthesia	Silk			Catgut		
	Total Cases	Suppurative Infections		Total Cases	Suppurative Infections	
		No.	Per Cent		No.	Per Cent
Local	557	8	1 43	85	2	2 35
General and spinal	727	10	1 37	878	25	2 84

* After Shambaugh, in *Surg., Gynec. & Obst.*, 64: 765, 1937.

TABLE IV

EFFECT OF SUTURE MATERIAL UPON THE INCIDENCE OF WOUND COMPLICATIONS*

Suture	Number of Operations	Suppurative Infections		Minor Wound Complications		Total Wound Complications	
		No.	Per Cent	No.	Per Cent	No.	Per Cent
Silk	1284	18	1 4	8	0 62	26	2 0
Catgut	963	27	2 8	51	5 29	78	8 1
Silk and catgut.	113	2	1.77	2	1 77	4	3 0
Totals	2360	47	1 99	61	2 59	108	4 57

TABLE V

EFFECT OF SUTURE MATERIAL UPON HEALING OF SUPPURATIVE WOUND INFECTIONS*

	Number of Cases	Average Hospital Convalescence, Days	Average Total Convalescence, Days
Silk, slight suppuration..	5	17	25
Catgut, slight suppuration	12	19	24
Silk, marked suppuration	13	22	42
Catgut, marked suppuration ...	14	26	46

frequently in the catgut series. (Tables IV and V.)

I quote Dr. Shambaugh's conclusions,

THE PRINCIPLE OF SILK TECHNIQUE

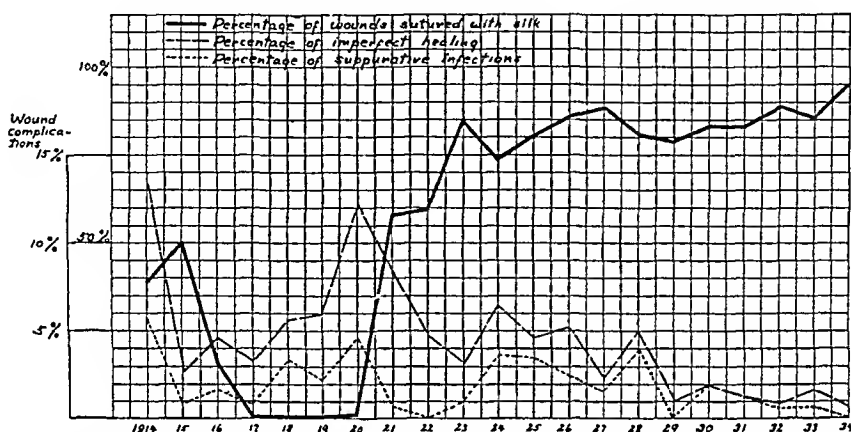The following are some of the principles involved in the silk technique:

FIG. 1. Graph showing relationship between the employment of silk and the annual incidence of wound complications. (From Shambaugh, in *Surg., Gynec. & Obst.*, 64: 766, 1937.)

because they represent the impressions formed by those who have used silk in a large number of cases:

"1. The alcohol and bichloride method of skin preparation compares favorably with other methods as regards the incidence of postoperative wound infection.

"2. Suppuration in clean operative wounds is more likely to occur in elderly patients than in the younger group.

"3. Anesthesia by local injection does not increase the likelihood of wound infection.

"4. In a controlled series the incidence of suppurative wound infections where catgut was employed as a suture material was twice as great as where silk was used.

"5. Where fine silk (No. 4) is used and the principles laid down by Halsted are followed, the presence of silk in suppurating wounds does not, on the average, delay the healing of the wound.

"6. Infected wounds may heal completely and permanently without discharging the silk sutures.

"7. The presence of fine silk in infected wounds may delay healing for periods up to three or four months, but this is exceptional and should not deter one from employing the silk technique in suitable cases."

No. 4 black twisted silk is ordinarily used for most purposes, except in the fascia where a larger size up to No. 9 may be used. The silk should first be dipped in melted bone-wax. The excess wax is wiped off and the silk is autoclaved. It should not be sterilized several times, as it soon loses its strength from repeated sterilizations. A small half-circle No. 2 or 3 French needle is used, except in intestinal work where a straight needle is usually preferable. The sutures are usually cut about 18 inches long. It is best not to use the same suture more than once in a French needle as it may become frayed. A small needle holder must be used, and it is best to clamp the French needle near its middle. The hemostat used for clamping the vessels must be small. One should attempt to clamp only the bleeders; if any tissue is included in the clamp it must be the smallest amount possible. The strangulation of large amounts of tissue must be avoided, but all bleeding must be controlled immediately. The silk suture, the finest that will furnish the necessary strength, must be cut at the knot. Silk sutures are usually interrupted, except in the peritoneum, where they may be continuous. The interrupted skin suture is the only one with which I am familiar

that can be removed without the possibility of contamination of the wound, that is, without drawing some of the exposed

with gauze. Silver foil is used to cover the incision and it helps to prevent contamination of the wound.
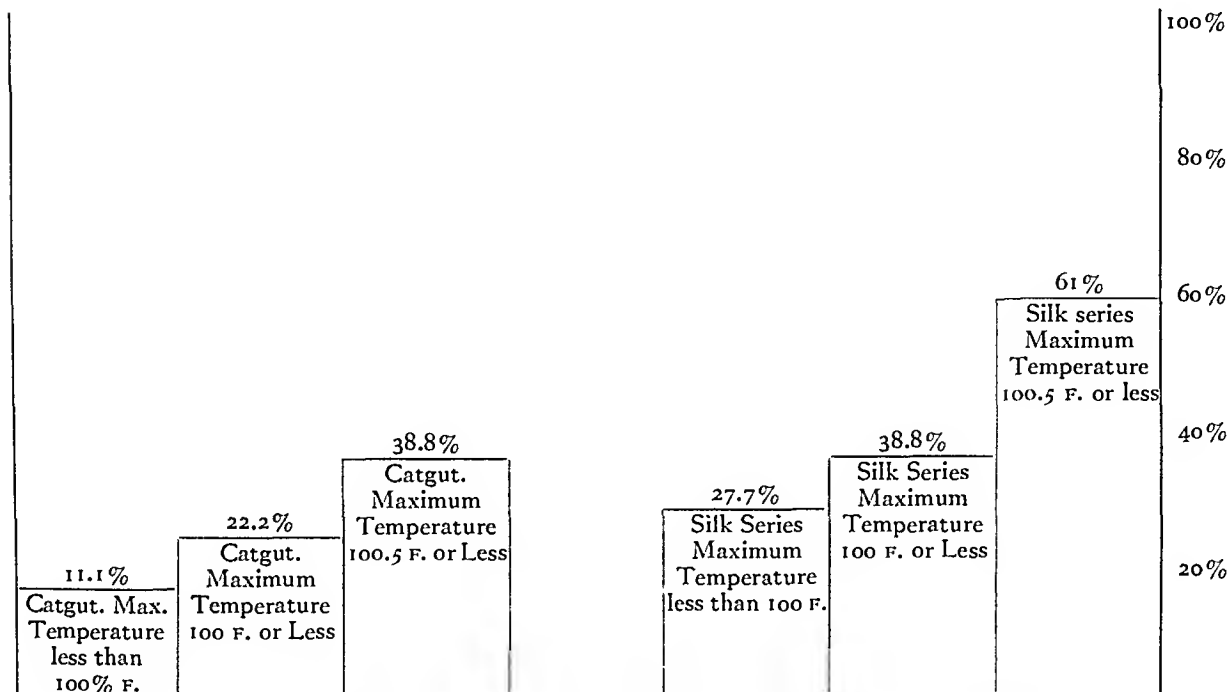

FIG. 2. Graph showing the comparison of temperatures in a series of catgut and silk cases.

part of the suture through the tissues. Skin sutures should be just tight enough to approximate the skin edges; they should not be under tension. A subcutaneous suture is used for the purpose of almost approximating the skin edges, and should be placed so the knot will be underneath. The bite should include only the deeper layer of the skin and should be taken a short distance from the edge of the wound. This subcutaneous suture is very important, because it keeps tension off the skin sutures, helps to produce a narrower scar, and closes dead space. All infections of operative wounds do not occur during the operation; a fairly large percentage occurs afterward from contamination due to carelessness in changing dressings, from carelessness in removing properly placed sutures, and from removing improper sutures. In order to avoid trauma, incisions should be of sufficient length to give adequate exposure. The edges of wound and skin during operation should be protected

PRELIMINARY REPORT

I am well aware of the fact that the small number of cases in this preliminary report cannot be used for the purpose of forming definite conclusions. This report is a series of unselected and consecutive private cases to compare the difference in temperature, etc. when cat-gut and silk were used. In this report there are thirty-six cases, eighteen in each series. In a few cases in the silk series, catgut was used in the peritoneum in order not to delay the surgeon following; catgut was also used in the pelvic part of the operations and around the sites of the drainage wounds. In all cases the peritoneal cavity was opened; there were thirty-three intra-abdominal operations and three inguinal herniorraphies (two herniorraphies in the catgut series, and one in the silk series). Drainage was necessary in six cases: one cholecystectomy, one ruptured appendix, and one tubo-ovarian abscess in each series. There were three cases of acute appendicitis

in the silk series and none in the cat-gut series. There was one case in the silk series where appendectomy was done for chronic
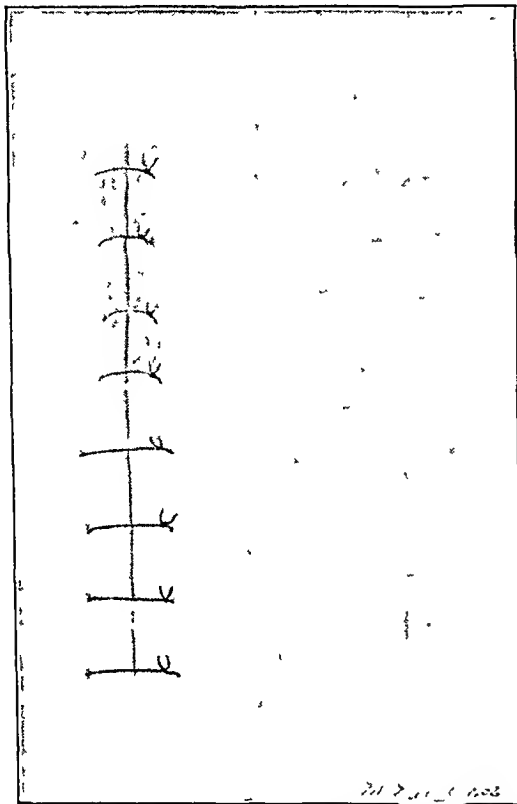

FIG. 3. Difference between the effects of tight and loose skin sutures. The cross scars are the result of anemic necrosis. (From Reid, in *South. M. J.*, 26: 110.)

appendicitis, and there were four such cases in the catgut series. The other operations included appendectomies for subacute appendicitis; supravaginal hysterectomies and various combinations of operations on the internal female organs, which were about equally divided between the two series. As the lowest temperatures are obtained in abdominal surgery in which there is simply an appendectomy for chronic appendicitis, and as there were no cases of acute appendicitis in the catgut series, it seems that if any particular difference existed in the mildness of the conditions, it was in favor of the catgut series.

As indicated by Figure 2, there were definitely lower temperatures in the silk series, but because of the small number of cases no definite conclusions can be reached; however, if this same advantage for silk could be maintained in a large series of cases it would be a most important point in favor of the silk technique.

In the catgut series 11.1 per cent had a maximum temperature of less than 100 F. In the silk series this was true in 27.7 per cent. In the catgut series the maximum temperature was 100 F. or less in 22.2 per cent; in the silk series in 38.8 per cent. A maximum temperature of 100.5 F was observed in 38.8 per cent of the catgut series and 61 per cent of the silk series.

If, when using the silk technique in clean cases, the postoperative temperature goes above 100.5 F., one should look for complications. They will not always be present, but with the silk technique temperatures are usually low, and a rise above 100.5 F. often indicates that something is wrong. The maximum temperature in the patient who had the least elevation of temperature in the catgut series was 99.8 F. The diagnosis was chronic appendicitis. The maximum temperature in the patient who had the least elevation of temperature in the silk series was 99.4 F. The diagnosis was chocolate cyst of the left ovary; cystic right ovary; chronic appendicitis, and partial stenosis of the cervical canal. There were three cases of acute appendicitis in the silk series and the maximum temperature did not reach 100 F. in any case.

No evidence of infection occurred in any of the clean cases in the silk series. In the drainage cases in the silk series there was no infection except around the drain, and the drainage wounds healed promptly with no evidence of infection in any other part of the incisions.

The duration of operations when using the silk technique is usually from twenty-five to fifty minutes longer than when using catgut, and because of the longer duration of the operations it is advisable to use basal anesthetics more often. In this series avertin was used as a basal anesthetic, in doses of 60 or 70 mg. per kilo of body

weight. Avertin was not used when there was a diseased condition of the biliary system or kidneys, or in hypotension.

The coöperation of the interns, anesthetist and operating room nurses is absolutely essential. The patient is the most important person in the operating room during the operation, and everyone connected with the operation should pay particular attention to the various steps so there will be no unnecessary delay.

CONCLUSIONS

From this preliminary report no definite conclusions can be reached in regard to the use of silk; however, it does seem that there is some justification for a favorable impression from the results obtained in the silk series. Certainly the lower the temperatures, the more comfortable the patients and the fewer the complications.

The milder postoperative courses observed when using the silk technique seem to result from a combination of circumstances, of which silk is the most important, as its use commands greater gentleness and more careful hemostasis.

While there were lower temperatures in the silk series, the things which impressed me most were the general well-being of the patients and a milder postoperative course than I have been able to obtain before. There also seemed to be less distention and fewer gas pains.

From the experience of those who have used the silk technique in a considerable number of cases and from my own limited experience, it appears that silk has a definite superiority over catgut in regard to wound complications.

Trauma, hemorrhage, and infection are the big factors in producing postoperative discomfort and complications.

REFERENCES

Beck, William C. Preparation of operative field, report of a survey of seventeen surgical teaching clinics. *Arch. Surg.*, 33: 876 (Nov.) 1936.

Cutler, Elliott C. Postoperative complications. *J. Michigan State Med. Soc.*, June 1928.

Cutler, E. C. Postoperative complications. *Proc. Inter-State Post-Graduate Medical Assembly of North America*, November 1934.

Cutler, E. C. The Changing trend in surgical technique. *Surg., Gynec. & Obst.*, 56: 963 (May) 1933.

Cutler, Elliott C., and Holloway, John W. Indispensable uses of narcotics in surgery. *J. A. M. A.*, 96: 857–859 (March 14) 1931.

Doane, Joseph C. Is standardization of surgical technique possible? *Mod. Hosp.* 36: 81 (Jan.) 1931.

Elmer, Walter G. Surgical technique. *Ann. Surg.*, 89: 328 (March) 1929.

Havens, Fred Z. Removal of calculi from the submaxillary ducts; a trick in tying a knot; a simplified method of local anesthesia for removal of keratomas and sebaceous cysts. *S. Clin. North America*, 15: 1229, 1935.

Jett, F. H. Operative infection. *J. Indiana State M. A.*, 29: 517 (Oct.) 1936.

Levi, Alexander A. Buried knot or day stitch. *Med. Rec.*, Aug., 1933.

Marshall, Harold K., and Thompson, Robert H. Mortality lessons in a series of 4,029 gynecological operations. *Calif. West. Med.*, 45: 263 (Sept.) 1936.

Reid, Mont R. A general consideration of blood supply in the practice of medicine and surgery. *South. M. J.*, 26: 107 (Feb.) 1933.

Shambaugh, Philip. Postoperative wound complications. *Surg., Gynec. & Obst.* 64: 765–771, 1937.

Shambaugh, Philip, and Dunphy, J. E. Postoperative wound infections and the use of silk: an experimental study. *Surgery*, 1: 379 (March) 1937.

Worrell, Ralph. A few surgical principles. *M. J. Australia*, 1: 256–258 (Feb.) 1931.

CASE REPORTS

INGUINAL HERNIA DUE TO DIRECT TRAUMA: TECHNIQUE OF OPERATION

GOLDER L. McWHORTER, M.D.

Late Associate Clinical Professor of Surgery, Rush Medical College, University of Chicago

CHICAGO, ILLINOIS

INGUINAL hernia as a result of direct accidental trauma is rare and the technique of repair must be adjusted to the extent of laceration of the tissues.

Traumatic hernia may be a result of either direct or indirect trauma. It is usually medial, but may be lateral to the inferior epigastric artery or involve both regions. Hernia resulting from direct trauma may be due to laceration of the protecting structures, in an accident, at operation or from intentional self injury. Hernia from indirect trauma may result from an increased abdominal pressure with a predisposing patent peritoneal process within the cord or to a congenital or acquired weakness of the protecting muscles and fascia.

Erdman[1] stated that the enlargement of the external ring which was present in 2.2 per cent of drafted men may be a determining factor in direct hernia. A definite history of accidental indirect trauma was obtained by Fallis[2] in 62.1 per cent of 994 patients with hernia. Gorton[3] believed that the hernia of weakness in the absence of a preformed sac was rare. Moorhead[4] concluded that a hernia cannot develop in the absence of a preformed sac, a congenital predisposition or an acquired anatomic defect, but that trauma can aggravate, accelerate, accentuate, incréase or modify it. St. Jacques[5] believed that hernia following a direct blow to the abdomen was rare, but conceded that its sudden development following effort should be considered as distinct from the aggravation of a preëxist-ing one. The type of hernia is frequently difficult to diagnose until the relation to the inferior epigastric artery or the cord has been determined and is frequently a complicated or pantaloon type of sac.

Bull and Coley,[6] after investigating the alleged relation of injury to hernia in 10,000 cases, considered that only two were due to direct violence. Selby[7] reported a case of direct hernia following a direct injury in which operation disclosed a tear in the external oblique fascia, the internal oblique and the transversalis muscles.

Holland[8] has submitted six characteristic points for the diagnosis of traumatic hernia: immediate descent of the hernia; severe pain in the hernial region; marked prostration; symptoms of such severity that attention is called to it within twenty-four hours; absence of a history of a previously existing hernia; and evidence that the trauma or strain was adequate. Following direct accidental traumatic hernia there is usually visible evidence of trauma, such as superficial abrasions, edema, subcutaneous ecchymosis, hematoma, redness or increased warmth to the area.

REPORT OF CASE

B. P., a male of 22 years, entered the hospital August 20, 1936. He complained of a mass in the left groin which developed immediately upon being struck there by the handlebar of his motorcycle when he ran into a tree and was thrown to the ground.

When he stood up the pain was so severe in his left inguinal region that he had to lie down.

In a short time he was able to get up and he noticed a mass in his left groin. The next day the mass became smaller when he was lying of the pubis and laterally to within about 1 inch of the anterior superior spine of the ilium. This mass could not be reduced in size by
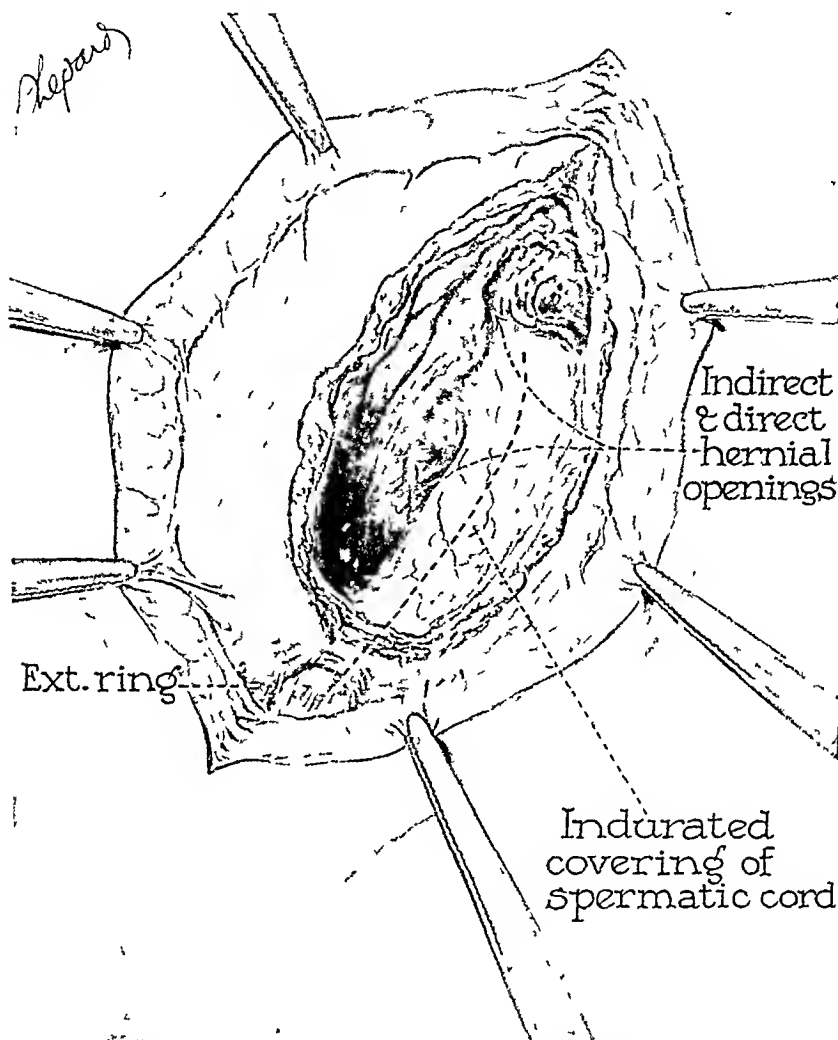

FIG. 1. Direct and indirect hernia due to extensive tearing of the fascia and muscles both medial and lateral to the inferior epigastric artery.

down and could then be pushed back, but it recurred upon standing. The patient was examined by a local physician thirty-four hours after the accident. He was unable to work but could walk about his home. One week after the injury the swelling did not subside upon lying down and he could no longer push it back.

Three months before the accident the patient had been examined by the physician at the factory where he worked and no hernia was discovered.

A sharply outlined mass could be noted on the left side, limited below by Poupart's ligament. It extended medially to the tubercle

pressure. There was an indurated, slightly discolored area in the skin just lateral to the pubic spine, indicating the site of a recent injury, and below this an area of subcutaneous ecchymosis. The swelling extended $2\frac{3}{4}$ inches above and $3\frac{3}{4}$ inches parallel to Poupart's ligament. It was elevated about 1 inch, while the superior and lateral borders were less abrupt than that above Poupart's ligament. The external inguinal ring, which was just medial to the mass, admitted only the tip of the index finger. Both testicles were equal in size with no evidence of injury. Peristalsis was heard distinctly over the swelling and there was definite bulging upon coughing.

At operation on August 24, 1936, a complete tear was found in the external oblique fascia above and extending laterally a short distance

The conjoined tendon was sutured with chromic catgut to Cooper's ligament along the ileopectineal line and the internal oblique and
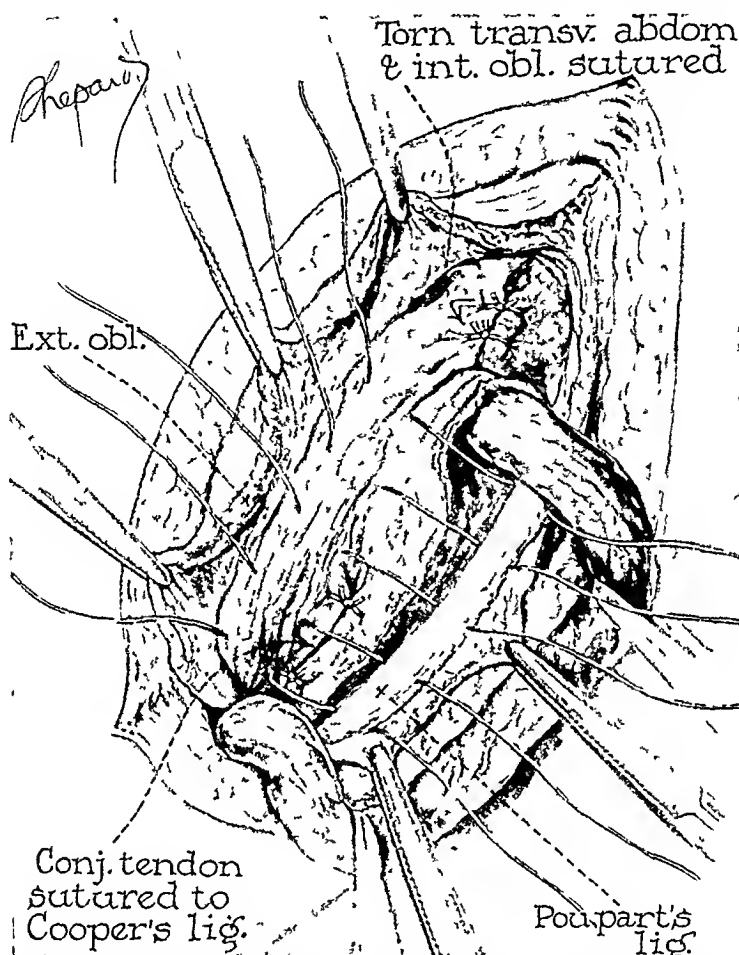

FIG. 2. Conjoined tendon sutured to Cooper's ligament. Muscles and fascia repaired and sutured to Poupart's ligament beneath the cord. Constriction avoided by short vertical incision in underlying fascia at new exit.

beyond the inguinal canal, except for a few fibers forming the external ring. The underlying internal oblique and transversalis muscles and the deep fascia were torn through, producing a defect in Hesselbach's triangle 1 by 2 inches in extent, extending laterally beyond the inferior epigastric artery $1\frac{1}{2}$ inches and surrounded by about 30 c.c. of old blood serum. The torn muscle fibers were indurated and the cord which extended below the hernia was covered with a dense layer of fibrin (Fig. 1.) The torn structures were identified medial and lateral to the inferior epigastric vessels, but due to the induration it was impossible to suture them to Poupart's ligament at its medial end.

transversalis muscles to the outer portion of Poupart's ligament beneath the cord. The defect in the muscles lateral to the inferior epigastric vessels was repaired by suturing the ends of the transversalis and the internal oblique muscles together with mattress sutures. (Fig. 2.) The edges of the torn external oblique fascia were sutured together beneath the cord with imbrication but without constriction by making a short vertical incision in the underlying edge of fascia. The subcutaneous fascia and skin were sutured over the cord. The patient was discharged without complications on the fourteenth day.

Examination six months later showed a firm abdominal wall although the patient had been

doing heavy labor for the preceding four months.

REFERENCES

1. Erdman, S. In Nelson Loose Leaf Surgery, 4: 610 Thomas Nelson and Sons, New York.
2. Fallis, L. S. Inguinal hernia: 1600 operations in ten years. *Ann. Surg.*, 104: 403 (Sept.) 1936.
3. Gorton, H. W. Compensable hernia. *J. Indiana M. A.*, 23: 521 (Nov.) 1930.
4. Moorhead, J. J. Traumatic Surgery, 2nd ed. Philadelphia, W. B. Saunders Co.; Relation of trauma to hernia. *New England J. Med.*, 209: 568 (Sept. 21) 1933.
5. St. Jacques, E. Accidental hernias and their compensation. *Canad. M. A. J.*, 22: 661 (May) 1930.
6. Bull and Coley. Quoted by Moorhead.[4]
7. Selby, C. D. Direct abdominal hernia of traumatic origin. *J. A. M. A.*, 47: 1485, 1906.
8. Holland, J. H. *New England J. Med.*, 209: 579 (Sept. 21) 1933.

The outstanding facts concerning the effects of pituitary extract on the metabolism of fat appear to be (1) large doses of anterior pituitary extract bring about a deposition of fat in the liver . . . —and (2) produce ketosis which is probably associated.

From—"The Physiology and Pharmacology of the Pituitary Body," vol. 2, by H. B. Van Dyke (University of Chicago Press).

RESECTION OF CARCINOMA (CARCINOID?) OF THE INFRAPAPILLARY PORTION OF THE DUODENUM INVOLVING THE AMPULLA OF VATER*

REIMPLANTATION OF THE COMMON AND PANCREATIC DUCTS

ALEXANDER BRUNSCHWIG, M.D. AND ALICE CHILDS, M.D.

Associate Professor of Surgery and Attending Surgeon, University of Chicago Clinics

Assistant, Department of Medicine, University of Chicago Clinics

CHICAGO, ILLINOIS

JUDGING from the number of cases reported in the literature carcinoma of that portion of the duodenum distal to the ampulla of Vater is quite rare. Lieber, Stewart and Lund have recently reviewed the literature on this subject and of sixty-two cases recorded, only twenty-eight reports were considered sufficiently descriptive to be acceptable as instances of this condition. To this series they added two cases observed among 23,500 autopsy records in Philadelphia. Of twenty-one patients operated upon, thirteen died within fifteen days of the operation of complications directly associated with the surgical intervention. Of the remaining eight, five died within two to three months, two were reported alive three months after operation and in one no postoperative details are given.

Some of the procedures followed were: gastroenterostomy in twelve cases (accompanied by cholecystogastrostomy or duodenostomy in two instances); cholecystostomy alone in one case; and resection with anastomosis in six cases. Of the latter group two were alive after three months and a third died three months after the operation.

Because certain conditions are of such rarity that it falls within the province of most individual surgeons to meet with them perhaps only once or twice over a span of many years, it is felt that individual reports of such conditions are justified. In this manner a composite record of experiences may be compiled. For these reasons the following case is recorded:

R. H., white female of 41, married, was admitted to the University of Chicago Clinics on the medical service of Dr. George F. Dick October 22, 1937. For the previous two years the patient had been aware of increasing fatigability to the point where by 10 A.M. it was almost impossible for her to continue with her domestic work which she had been able to do without difficulty. No palpitation, shortness of breath, edema about the ankles, or tremor was discernible.

Synchronous with the above symptoms there appeared to be an increase in quantity of stool and irregular periods of frequent stools. On one or two days there would be five to seven mushy stools and lower abdominal pains followed by two to four days when there would be only one stool. The character of the stools had also changed; they were very "sour" and foul and varied from clay-colored to dark brown. No gross blood or tarry stools were ever noted. The appetite remained good, but during the two years the patient had lost ten pounds. No history of icterus was given. The family history was irrelevant. Four children were living and well.

Physical examination revealed as well developed, slender, middle aged white female, not acutely ill. Peristaltic waves about the umbilicus were visible, but for the patient's weight (96 pounds) this did not appear necessarily abnormal. To the right and slightly above the umbilicus a vague deep mass about the size of an egg could be palpated sometimes and when it was felt the patient complained of distinct tenderness. No cervical or inguinal

adenopathy and no icterus were present. The physical findings were otherwise negative.

Blood pressure was 108/70, pulse 78, temperature 98.6°F. The Wassermann and Kahn

fluid was present. The gall-bladder was markedly distended by bile but its wall appeared normal; no stones were palpable. Exploration

Fig. 1. Roentgenogram showing large filling defect in duodenum due to neoplasm in lower second portion. s, stomach. d, duodenal bulb.

perature 98.6°F. The Wassermann and Kahn tests were negative. Red blood cells numbered 4,200,000 and the white 13,000. Hemoglobin was 82 per cent. A differential count showed 73 per cent polys, 21 lymphocytes, 3 monocytes, and 1.2 per cent reticulocytes. Urinalysis was negative. Multiple stool examinations showed light to clay-colored stools very foul, and of sour odor, containing much fat; occult blood was also present. The sugar tolerance curve was normal and the basal metabolic rate minus 10. Free acid was present in the Ewald meal. Blood chemistry showed: 176 mg. per cent cholesterol; 9.7 mg. per cent serum calcium; 4.4 mg. per cent serum phosphate.

Roentgenographic examination revealed a tumor mass approximately 8 by 5 cm. lying within the distended lumen of the second portion of the duodenum. The colon, stomach and esophagus were negative.

The preoperative clinical diagnosis was a neoplasm of the pancreas or duodenum with partial obstruction of the pancreatic ducts.

Operation was done under spinal anesthesia on November 26, 1937. The abdomen was entered through a high midline incision. No free
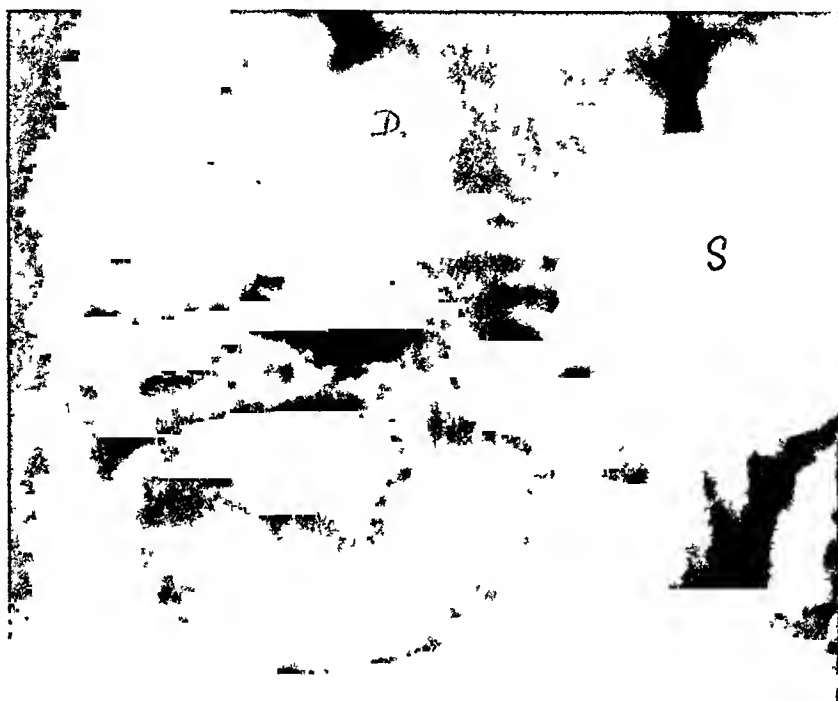
was negative except for an oval tumor mass in the lower part of the descending duodenum. The latter was quite mobile. The transverse colon and mesocolon were lifted upward and covered with moist towels. The small intestines were packed downward and to the left in a similar manner. With the tumor bearing segment of duodenum immobilized by the left band a longitudinal incision 8 cm. long was made through the anterior duodenal wall over the tumor which appeared to arise from the posterior wall. When this wound was spread, a lobulated grey tumor mass with several shallow ulcerations upon it was exposed. Palpation revealed it to be quite firm—circumscribed and movable in the duodenal wall. The gross characteristics were those of benign tumor. It was felt at the moment that the neoplasm was essentially submucosal; when pulled upward it appeared to have a thin elongated pedicle of mucosa. There was no evidence of infiltration about the base. Inspection of the duodenum above the tumor failed to reveal the ampulla of Vater. The tumor was pulled upward again and with scissors dissection was "shelled out" by cutting transversely through the pedicle from

left to right. When the last 1 or 2 cm. of the pedicle were cut, a small gush of clear fluid was noted and then a vigorous gush of bile. The latter was sponged and its source revealed as

peritoneum closed over this in a separate layer. Palpation of the now relaxed and partially filled gall-bladder again revealed no stones. The abdominal wound was closed in layers.
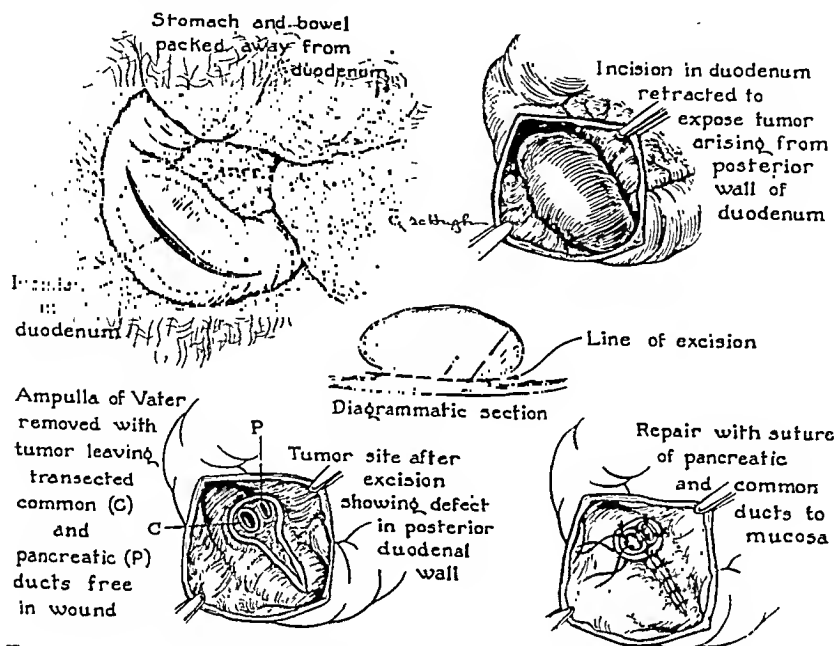

FIG. 2. Diagrammatic illustrations of operative removal of tumor in second portion of duodenum.

the transected common duct lying free in the right portion of the elongated narrow defect in the posterior duodenal wall resulting from the excision. A small suction tube was inserted into the duct which kept the field dry of bile. Just above the common duct, also lying free in the wound, the thin walled duct of Wirsung was identified. Both ducts were then immobilized by clamping of their edges with mosquito forceps, care being taken to include only a minimum of tissue.

As stated, it was the impression at this time that the muscularis of the posterior duodenal wall had not been completely transected, and because the excised base of the pedicle of the tumor left but a narrow elongated wound no probing through the latter was performed, there being no gross evidence of remaining neoplasm. Using fine "nerve silk" the common and pancreatic ducts were sutured together by two interrupted stitches and in a similar manner sutured to the surrounding mucosa. The remainder of the wound was closed by a single layer of interrupted silk sutures of heavier grade. The linear incision through the anterior duodenal wall was closed by two rows of interrupted silk sutures and the posterior parietal

There was a minimal reaction after operation, the highest temperature being 100.4°F. on the second day.

Following operation there was, of course, little change in the character of the stools. The patient was followed in the Out-Patient Department and by February 18, 1938, had gained 10 pounds and felt generally much improved. She was eating liberally of a general diet. Fatty stools continued. When the patient was last seen in February, 1939 the improvement had been maintained and the only complaint was an excessively fatty stool every three to four weeks.

Histologic Study. A section through the neoplasm, including overlying duodenal mucosa, showed atrophic and degenerative changes in the latter due to pressure from the neoplasm below. The tumor itself was composed of anastomosing broad cords of small, closely packed, polyhedral or cuboidal cells with rather dense eosinophilic cytoplasm and large rounded nucleus rich in finely divided chromatin. In some areas the cells were grouped about a lumen, forming alveolae. Only an occasional mitotic figure was present. Between the masses and cords of neoplastic cells were bands of

smooth muscle—the muscularis of the duodenum. The extent of the latter in the sections made it highly probable that the tumor had infiltrated through the entire duodenal wall
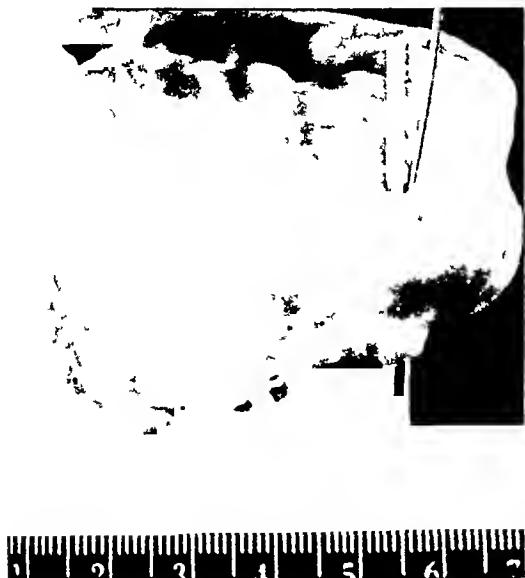

FIG. 3. Gross appearance of excised neoplasm. The metal probe is in the main pancreatic duct and the wooden probe in the common bile duct.

and was not essentially within the submucosa as was thought to be the case at operation.

The impression gained upon study of the routine sections in conjunction with Dr. Eleanor Humphreys of the Department of Pathology was that the tumor resembled more a carcinoid tumor than any other type of epithelial neoplasm and that it was not typical of any of the usual varieties of carcinoma of the bowel. Sections were stained for argentaffine cells by Dr. Humphreys but none was found. Mucicarmine stains revealed no mucus in the cells.

The pathologic diagnosis was carcinoma, but quite possibly carcinoid tumor, of the second portion of the duodenum, with involvement of the ampulla of Vater.

DISCUSSION

1. *The Histologic Character of the Tumor.* An absolute diagnosis of carcinoid could not be made in the absence of argentaffine granules, although, as stated, the hematoxylin and eosin sections strongly suggested this type of neoplasm. The tumor in all probability was a slowly growing malignant one. From the history it had certainly been present for more than two

years. It is generally held that carcinoids, especially those not in the appendix, are of low grade malignancy and, if not removed, eventually produce metastases.

FIG. 4. Photomicrograph (× 60) showing neoplasm to be composed of small closely packed epithelial cells.

2. *The Clinical History.* Carcinomas of the ampulla or of the duodenum immediately adjacent to it usually produce obstructive jaundice which is generally considered a cardinal clinical sign of ampulla tumors. In this case the clinical picture was essentially one of steatorrhea due to slowly increasing obstruction of the main pancreatic duct. There was also, of course, encroachment upon the common bile duct, which in this case appeared in the ampulla to empty separately into the duodenum. No clinical evidence of biliary obstruction was observed. It would appear from the findings and the history that the neoplasm began in the duodenal wall a short distance below the ampulla of Vater and involved the latter during subsequent extension.

3. *The Effects upon the Pancreas.* It has been well established that in experimental animals ligation of the pancreatic duct is followed, among other things, by degeneration of acinar tissue of the pancreas. While in the above case pancreatic tissue was not removed, there was in all probability extensive degeneration of the pancreas. Because of this it is to be expected that steatorrhea would continue for some time, until sufficient acinar tissue has reformed

to supply adequate pancreatic juice for normal digestion.

It would appear that Halsted in 1898 was the first successfully to remove a portion of the duodenum and reimplant the common and pancreatic ducts. The patient lived seven months and died from recurrence of the lesion.

In a review of the recorded instances of radical operations for carcinoma of the periampullary region of the duodenum from 1925 to 1934, Hunt and Budd report four deaths in eighteen cases or a mortality of 22 per cent.[2] A review by Cohen and Colp[1] of cases previously reported in the literature showed a gross mortality of 43 per cent in fifty-eight instances where a variety of procedures was followed.

SUMMARY

An instance of a large, slowly growing carcinoma (carcinoid?) of the second por-

tion of the duodenum secondarily involving the ampulla of Vater is recorded. Transduodenal resection and reimplantation of common and main pancreatic ducts were performed. The patient remained free from evidence of recurrence fifteen months after operation.

The special clinical feature of the case was that steatorrhea due to obstruction of the pancreatic duct was the outstanding sign and no clinical evidence of common duct obstruction was present.

REFERENCES

1. COHEN, I., and COLP, R. Cancer of the periampullary portion of the duodenum. *Surg., Gynec. & Obst.*, 45: 332, 1927.
2. HUNT, V. C., and BUDD, J. W. Transduodenal resection of the ampulla of Vater, etc. *Surg., Gynec. & Obst.*, 61: 651, 1935.
3. LIEBER, M., STEWART, H. L., and LUND, H. Carcinoma of the infrapapillary portion of the duodenum. *Arch. Surg.*, 35: 268, 1937.

OSTEOGENIC SARCOMA ARISING IN A DEVITALIZED PART FOLLOWING INJURY

I. Arthur Stoloff, m.d.

Adjunct Professor of Surgery, New York Polyclinic Medical School and Hospital; Attending Traumatic Surgeon
Beth David Hospital

NEW YORK CITY

AT the present time the etiology of sarcoma is still surrounded by mystery. The theory of chronic irritation holds weight as a probable factor in many anatomic sites, but, whether the injury of a single trauma is sufficient to induce tissue neoplasia is still a very moot question, and one which frequently baffles the surgeon engaged in traumatic and industrial surgery.

The case herein presented is of clinical and legal interest because the relationship of trauma to tumor was complicated by the fact that the part had suffered devitalization for many years. Although medical testimony was controversial, legally the case was upheld as compensable.

Ewing[1] is of the opinion that chronic irritation is the type of injury that may be a factor in the production of malignant neoplasia.

William B. Coley,[2] in the year 1897, presented a paper before the New York Surgical Society, entitled, "The Influence of Injury upon the Development of Sarcoma," in which he analyzed 170 cases of sarcoma which were personally observed by him, forty-six of which gave a definite history of previous local injury. In 1910, in another publication,[3] he reported 970 cases of sarcoma personally observed, in which there was a history of previous local trauma in 225 cases, or 23 per cent; and 250 cases of carcinoma in which there was a history of injury in eighty-two cases, or 32.8 per cent.

Coley and Higinbotham[4] in a recent article contend that there is a direct relationship between trauma and malignant tumor based upon a hypothetical micro-organism theory. They state: "We may assume that malignant tumors in man are due to a micro-organism that is latent in the circulation, and which gives rise to symptoms of malignancy only after the normal resistance of the cells is broken down, in some instances by local trauma. The micro-organism thus finds a suitable soil in the damaged cell, forming a symbiosis with the cell and causing a proliferation and multiplication resulting in a malignant tumor."

Contrary to this belief, Knox,[5] in 1929, definitely ruled out trauma as an etiologic source of tumors. She placed considerable weight upon the inability of a single trauma to produce tumors experimentally. Lubarsch and Ribbert were both unsuccessful in producing neoplasia, whether by a simple traumatization of tissue or by the injection of traumatized tissue into the animals. Knox reasoned that as a result of a single trauma scars may persist as demonstrable lesions either because of the presence of foreign material implanted in the beginning, or possibly resulting from an infection. Therefore, the important factor seems to be that of chronic irritation rather than acute single trauma. Such a sequence would obtain support in the report of Bloodgood,[6] who found that of forty-eight sarcomas of the skin, the neoplasms developed in a nevus eight times, in a scar twenty times, and in a fibroma nine times.

Lewy[7] studied 23,389 injuries in one year and found only thirty-seven tumors. From this extensive review he believes that trauma is not a primary cause of tumor.

William Seaman Bainbridge[8] writes that it is conceded that surgeons with the full facts assembled before them—clinical his-

tory, signs, symptoms and laboratory reports—are the ones to give the decision in any case as to whether the neoplasm

FIG. 1. Appearance of patient before amputation, showing marked edema and deformity of left arm.

which follows the trauma is the direct result of the injury. Similarly, Kaplan and Rubenfeld,[9] in discussing soft tissue sarcoma, state: "In our opinion, based entirely upon clinical factors, the patient, the tumor, and the traumatic effect must, in every instance be individualized, since the sequence is apparent in some cases and dubious in others."

CASE REPORT

A. K., 25 years old, a white male, presented himself for examination, on April 10, 1934, with a history of having fallen from a wagon on February 5, 1934. He struck the anterior surface of the lower third of his left arm, at which site there was enlargement, deformity and mass in the soft tissue suggestive of an encapsulated hematoma. Two weeks after the original injury, he was referred to the Post-Graduate Hospital, where he was x-rayed, but no treatment was instituted since operation was advised and refused.

I saw the patient nine weeks after the injury. A scar from the left scapula to the elbow joint remained as a result of a previous operation during childhood. (Fig. 1.) A plastic operation had been done in childhood for web fingers, with a resultant lymphangitis. The entire upper extremity was edematous and enlarged to about twice the size of the other. There was a circumscribed mass about 1 inch above the wrist, hard in consistency, and apparently attached to the bone. The area about the mass was inflamed and fluctuant.

X-rays showed extensive involvement of the distal half of the radius, with roughening of the periosteum and irregular decalcification. The principal changes were in the anterior medullary portion. There was an opaque mass 2 cm. distal from the bone, which might be a bony new growth and had the characteristics of an osteogenic sarcoma. (Fig. 2.)

After consultation it was decided to do a biopsy. Accordingly, on April 16, 1934, under local anesthesia, an incision was made through the skin and superficial fascia, and a great deal of fibrous tissue exposed. A portion of this mass was explored; it extended to the fascia between the radius and the ulna and was at-

tached to both bones. The major portion of the mass was enucleated and the periosteum of the bone was thus exposed. A sharp spicule of bone, which was protruding, was removed.

cell tumors without definite bone formation yielded to persistent moderated and graduated doses of x-ray. It was his opinion that amputation was not justified and should be delayed
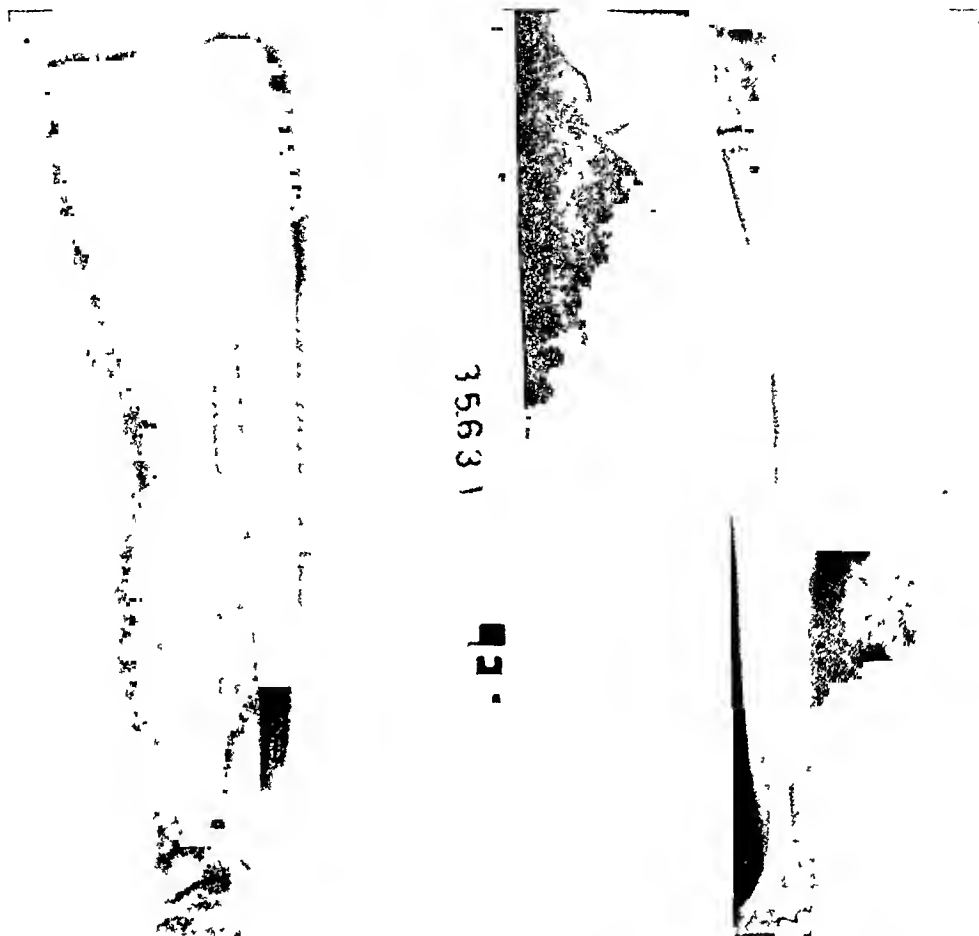

FIG. 2. Active destruction of the cortex of the distal third of shaft of the radius with no evidences of bone reaction here. There is, however, a discrete mass of new bone formation entirely separated from the cortex but within the tumor mass, probably due to secondary destruction.

Histologic examination showed a tumor consisting chiefly of fibrous spindle cells with multiple areas of necrosis. There were irregular fragments of bony tissue surrounded by fibrous tissue with evidence of necrosis. In one area of the fibrous tissue, there were a number of small spindle cells, which were moderately well differentiated but presented sarcomatous characteristics. The diagnosis of osteogenic sarcoma was given. (Fig. 3.)

Dr. Ewing was kind enough to reexamine the tissue and agreed with the diagnosis. He also added that while some of the spindle cells were extraperiosteal in location, he thought that the growth was periosteal in origin. He advised Roentgen treatment because, while the tumor itself was radioresistant, very frequently small-

until every method of conservative therapy had been instituted.

Dr. Alexander Fraser considered amputation the most feasible form of therapy since he felt that the growth was too highly differentiated to be sufficiently radiosensitive.

However, the plan suggested by Ewing was followed and the lesion was irradiated for several weeks, during which time reactions were carefully noted. There was no appreciable improvement in the neoplasm and amputation of the arm gave the only possible chance for cure.

On June 19, 1934, a high amputation of the left arm was done; the patient and his family were averse to a disarticulation. (Fig. 4.)

The patient made an uneventful recovery.

X-rays of the chest on August 15, 1934 were negative for metastasis. The patient was treated routinely until the wound was com-
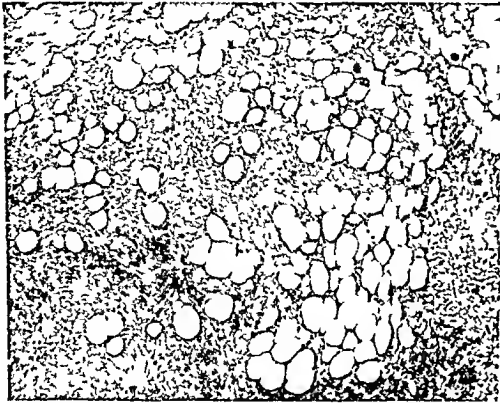

FIG. 3. Microsopic examination shows irregular fragments of bony tissue, surrounded by fibrous tissue, and evidence of necrosis. In an area in the fibrous tissue, there are a number of small spindle cells which are moderately well differentiated but present sarcomatous characteristics.

pletely healed. He had gained more than 35 pounds about one year after amputation.

On January 2, 1936 the patient returned, complaining of pain in the chest, coughing and shortness of breath, all of three weeks' duration. X-ray examination disclosed massive pleural effusion on the left side, displacing the heart, trachea and mediastinum into the right chest.

On January 14, aspiration of the pleural cavity was performed, and about 600 c.c. of bloody fluid was removed. A histologic examination of the fluid revealed a hemorrhagic pleural exudate negative for tumor cells.

Aspiration was done five times, and the histologic reports at all times revealed a hemorrhagic pleural exudate without tumor cells or any identifying organism.

The patient was discharged from the hospital, and two later thorocenteses obtained fluid of the same character.

Coley's serum and additional x-ray therapy to the stump and lungs were instituted. At this time, metastatic deposits appeared disseminated in the scalp.

The patient finally succumbed on July 8, 1936, about two and one-half years after the original injury.

DISCUSSION

The conditions first propounded by Segond in Paris, and accepted by the courts and compensation bureaus in Europe and America that must be present to establish the relationship between an antecedent
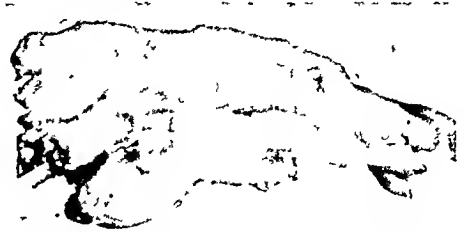

FIG. 4. Gross specimen of amputated forearm. Near the area of ulceration there is invasion of fat, skin and tendon. There is a dense tumor nodule penetrating almost the entire thickness of the arm. Both the radius and ulna are smaller than normal. The radius is involved by the tumor and shows considerable destruction of the outer shell. There is definite marrow involvement. There are other small portions of bony spicules in some areas of the tumor.

local trauma and subsequent development of a tumor, are: (a) the authenticity of the trauma; (b) sufficient importance or severity of the trauma; (c) reasonable evidence of the integrity of the body part prior to the injury; (d) correspondence of the tumor to the site of the injury; (e) date of appearance of the tumor not too remote from the time of the accident to be reasonably associated with it; (f) diagnosis established by clinical and roentgenologic evidence, supported if possible by microscopic examination.

We feel that the case reported is of special interest because of the hypothetical problems involved, opening fields for discussion of a practical nature. The six postulates listed above are generally accepted as formidable criteria. Evidence of integrity of the part prior to the injury is the question frequently productive of controversy in legal matters. The testimony must prove or disprove the presence of a neoplasm prior to injury. Certainly, a preëxisting tumor may be aggravated to more accelerated growth by trauma. Will an injury be adequate to promote malignant neoplasia in a part that has been devitalized over a period of many years?

We find no reference in the literature from which we may quote opinion. The case presented here gives substantial evidence of a part where the integrity had been disturbed for many years with old infection, lymphedema and stasis. I feel that severe trauma on this previously devitalized part was the immediate cause initiating a series of cellular changes which eventually led to a malignant bone tumor.

It is not amiss to suggest that future cases should be investigated more carefully to determine the integrity of the part. Careful history may yield clues of previous infections, injuries or degenerative processes which may have materially disturbed the integrity of the tissues.

SUMMARY AND CONCLUSION

This is a case of a young man, previously in excellent health, except for a congenital deformity of the left arm, forearm and hand, who sustained a direct injury to the arm and forearm. Two weeks after, a neoplasm of the forearm was found, which, upon histologic examination, proved to be osteogenic sarcoma. Radiation therapy proved futile, and was followed by a high amputation of the arm. Eighteen months later, metastases to the chest appeared. Death occurred two and one-half years after the time of injury.

I am of the opinion that there was a direct relationship between the injury and the subsequent development of the sarcoma.

It is worthy of mention that the causal relationship was established and upheld by the medical division of the New York State Department of Labor.

REFERENCES

1. Ewing, J. Relation of trauma to malignant tumors. *Am. J. Surg.*, 40: 30–36, 1926.
2. Coley, W. B. Influence of injury upon the development of sarcoma. *Ann. Surg.*, 27: 259–284, 1898.
3. Coley, W. B. Injury as a causative factor in cancer. *Ann. Surg.*, 53: 449–488, 1911.
4. Coley, W. B., and Higinbotham, N. L. Injury as a causative factor in development of malignant tumors. *Ann. Surg.*, 98: 991–1012, 1933.
5. Knox, L. C. Trauma and tumors. *Arch. Path.*, 7: 274–309, 1929.
6. Bloodgood, J. C. Diagnosis and treatment of border line pathological lesions. *Surg., Gynec. & Obst.*, 18: 19–34, 1914.
7. Lewy, R. Cancer in its relationship to trauma. Medical Division Bulletin, No. 1, State of New York, Department of Labor, May, 1923.
8. Bainbridge, W. S. Role of trauma in production of cancer. *Med. Times & Long Island M. J.*, 5: 39, 1934.
9. Kaplan, I. I., and Rubenfeld, S. Sarcoma of soft tissue. *Am. J. Roentgenol.*, 37: 1, 53–67, 1937.

A CASE OF INTERSTITIAL HERNIA*

Abraham O. Wilensky, m.d.   and   Jules D. Gordon, m.d.

NEW YORK CITY

THIS report is prompted because of the relative infrequency of the condition to be described and the problem in diagnosis that confronted the members of both the medical and the surgical services who saw the case.

CASE REPORT

J. F., age 62, gravida 9, para 3, was admitted to the medical service on January 5, 1938, complaining of pain in the left iliac fossa of three days' duration. The patient had had an operation on the cervix in 1907, an appendectomy in 1910, fractures of both humerri sustained in an automobile accident, in 1928, a tonsillectomy in 1929, and a right herniorrhaphy in 1936. The previous history was otherwise essentially negative.

Three days before admission to the hospital, the patient took an alophen pill before retiring. On the following morning, she felt pain in the left lower quadrant which was not severe but was persistent and remained localized.

Examination demonstrated a mass shaped like a sausage in the left lower quadrant, parallel to Poupart's ligament. The mass was tender to touch. The general condition of the patient was excellent. The hemoglobin was 78 per cent; red blood cells numbered 3,750,000; the white blood cells numbered 9,200, of which 62 per cent were polymorphonuclears. Stool examinations were negative for blood. The urine was normal, the temperature and pulse not elevated, and x-ray and sigmoidoscopic studies negative. The original opinion of the visiting staff was that there was an intra-abdominal tumor, probably a neoplasm in the sigmoid.

On January 8, 1938, the patient was operated upon under spinal anesthesia. An oblique incision, parallel to the long axis of the mass, was made in the left lower quadrant (the operator feeling that the most likely diagnosis was a tumor in the abdominal wall). The fibers of the external oblique were divided directly over the palpable tumor. On retracting the edges of the external oblique incision, a banana-shaped mass, about 3 inches long, was found, lying below the external oblique and between it and the internal oblique. The mass was adherent to the surrounding structures and had a bluish-green color. It extended from the general neighborhood of the internal ring upwards and outwards. Tracing the mass downwards seemed to show it continuous with and disappearing into the internal ring. Splitting of the covering disclosed that it was a hernial sac containing adherent gangrenous omentum. The omentum was freed and was resected in healthy tissue, after which the sac was treated by high ligation and ablation, as is customary in a hernioplasty. The operation was completed by an Andrew Ferguson herniorrhaphy. No other sac running towards the labia or along the round ligament was noted.

The postoperative diagnosis was interstitial hernia.

The postoperative course was uneventful except for a small hematoma in the lower angle of the wound.

DISCUSSION

The generally accepted views regarding the etiology of interstitial hernias is that an ectopic testicle diverts the hernial sac from its normal descent into the scrotum. Because MacReady, Gobel, and Lower and Hicken report a majority of the collected cases in their series associated with an ectopic testicle, this must be considered as an important factor. That this is not the only factor, however, or even an essential one, is readily appreciated when one uncovers the numerous case reports of interstitial hernia in men with normally placed testicles, and also in women. In many of the reported cases of this type of hernia in males with normally placed testicles, a constricted external ring, congenital or acquired in origin, has been found. There is

also a case on record of a hydrocele in the Canal of Nuck, evidently the obstructing factor in the causation of the interstitial hernia. In our case, as in Lower and Hicken's two cases, no obstruction was encountered in the inguinal canal or at the external inguinal ring. Nevertheless, it appears to us that some obstructive factor must in large measure be responsible for the occurrence of this hernia.

Interstitial hernia is one of the three types of a group of abnormal hernias in the inguinal region known under the heading of interparietal hernia. The latter may be defined as a group of unusual inguinal hernias in which the sac has failed to follow the course of the spermatic cord and has spread itself out between the various layers of the abdominal parietes. The interstitial variety, with which we are chiefly concerned in this paper, includes those hernias in which the sac lies either between the transversalis fascia and transversalis muscle, or between the internal oblique muscle and the external oblique aponeurosis. There is still another type of interstitial hernia in which the sac lies within the fibers of the internal oblique muscles. This, however, is a very rare condition. Moynihan denies the existence of any variety of interstitial hernia other than that in which the sac is found between the oblique muscles. Authentic cases, however, of other varieties have been reported by Gobel and Coley and Sultan.

The most common variety of interstitial hernia, as mentioned previously, is that in which the sac lies between the two oblique muscles. The case here reported, those of Lower and Hicken and that of Biegler were in this category. The probable explanation for this is purely a physical one and lies in the greater potential space and loose connective tissue between the two oblique muscles.

Although Moynihan, Halstead and Watson contend that biolocular sacs must be present in these types of hernias, our case presented only one sac, and about one-third of the cases reported in the literature had monolocular sacs.

Intestinal obstruction is usually encountered as the outstanding symptom. There were ninety-seven incarcerations in Gobel's 115 cases. Intestinal obstruction was absent in the cases of Lower and Hicken, Biegler, and in our own. The sac in our case, however, despite the absence of obstructive symptoms, contained gangrenous omentum.

In Biegler's and in our own case, a palpable mass above Poupart's ligament was found. In one of Lower and Hicken's cases, there was a swelling above the external inguinal ring.

The diagnosis should not be difficult in those cases presenting obstructive symptoms associated with an abnormally placed testicle and a palpable mass above Poupart's ligament. In other cases without the above combination of symptoms and signs, the diagnosis may be difficult or impossible. In the presence of a mass with pain alone, a differential diagnosis between diverticulum of the intestine, neoplasm of the intestine, tumor of the abdominal wall and interstitial hernia may be difficult, as our experience in this case shows. The diagnosis, however, should be suspected if the opposite side presents a hernia of the usual variety or there is a history of a previous herniorrhaphy, even though the patient may present only a mass and pain in one of the lower quadrants. Biegler's patient presented an ordinary inguinal hernia on the opposite side and ours gave the history of a herniorrhaphy performed two years previously on the opposite side.

REFERENCES

1. Johnson, A. B. System of Surgery, iv: 61, 1916.
2. Callander, C. L. Surgical Anatomy. Philadelphia, 1934. Saunders.
3. Lower, W. E., and Hicken, N. F. Interparietal hernia. Ann. Surg., 94: 1070, 1931.
4. Biegler, S. K. Interstitial hernia. J. Michigan M. Soc., 34: 438–439, 1935.

ON EXTRARENAL RETROPERITONEAL TUMORS*

Heinrich L. Wehrbein

BROOKLYN, NEW YORK

SINCE reporting a case of ganglio-neuroma in 1929, which was retro-peritoneal and extrarenal, we have encountered five other tumors so characterized by their location. There were three neuroblastomata in children, one leiomyosarcoma in an elderly woman, and one metastatic tumor in a middle aged male which arose from a tiny embryoma of the testis.

In all cases urologic structures were involved and all cases except the second had complete preoperative urologic examinations. The diagnostic difficulties in the fourth and the fifth case deserve particular attention.

The surgical difficulties were all due to blood supply control. The aorta was intimately involved in the third case, so much so that removal was impossible. It was also involved in the fifth case, but not so intimately. The renal vessels were the main source of blood supply in the first and the fifth case making the operations fairly simple. In the second case branches from the internal iliac artery supplied the tumor. The vena cava was invaded in the fourth case, thereby causing inoperability.

The surgical approach was transperitoneal in the first three cases and retroperitoneal in the last two.

One of the patients died shortly after the operation and one died two months later. The other three are alive and apparently well today.

CASE REPORTS

CASE I. The patient was a female child, aged 4 months. She was brought to the hospital because of vomiting, loss of weight, and mass in the abdomen noticed for the preceding three weeks. A large, round hard mass was found in the left side of the abdomen, extending from the costal margin to the iliac crest, and from the midline to the loin. The mass was painless and movable.

Cystoscopy showed P.S.P. function from both sides in eight minutes. A left pyelogram showed displacement of the left ureter medially and the left kidney high up in the abdomen. The left kidney pelvis and the calices showed crowding. (Fig. 1.) The chest and the long bones were roentgenologically negative.

An operation for removal of the mass was undertaken. Through a T-shaped abdominal incision and a transperitoneal approach it was possible to free the tumor completely and to determine its attachment to the lower part of the left kidney capsule. Due to the blood supply it was necessary to remove the kidney along with the tumor.

On pathologic examination the tumor proved to be 9 cm. in each of its three diameters and to be attached to the lower and mesial surface of the kidney capsule. The kidney itself was not involved. The histologic report was as follows: "The tumor is divided into lobules of varying size and shape, separated by connective tissue bands. The cells resemble medullary neural epithelium; they are for the most part small and of varying shape. Many of them produce glial fibers and in some foci well formed rosettes are seen. Aside from the rosettes the general architecture is that of strands of primitive neural epithelium found in some teratomata." (Fig. 2.) A diagnosis of neuroblastoma was made.

The postoperative recovery of the patient was uneventful and the child is reported to be alive and well four years after operation.

CASE II. A female child, aged 18 months, was brought to the hospital with the chief complaint of a mass in the lower abdomen which had been noticed for eight months and which had been slowly increasing in size.

Examination revealed a large, hard, nodular tumor in the right hypogastrium. It was very slightly movable and not painful; on rectal examination it appeared fixed to the right

sacroiliac synchondrosis. Cystoscopy was not undertaken, because the tumor could be outlined so definitely by palpation. The preoperative diagnosis was retroperitoneal neurocytoma.

FIG. 1. Displacement of left ureter and left kidney.

The operative approach was through an incision slightly to the right of the midline. The posterior peritoneum was incised medially to the right ureter which was anterior to the tumor. Dissection was fairly easy at first; the external iliac vessels and the ureter were easily separated and the tumor was then found to emerge from the first sacral foramen and to derive its blood supply from the internal iliac or hypogastric artery. It was necessary to cut numerous vessels arising from this artery and also several nerve bundles which were thought to come from S 1 and 2. The operation was very bloody and removal of the tumor was not thought to be complete, small portions of it apparently remaining in the first sacral foramen.

The tumor measured 7 by 8 by 4 cm. and had a thin capsule. On cross section it showed grayish-white nodules in a dark red stroma.

Sections showed a tumor made up of small, deep staining cells, arranged in nests or lobules which were separated by a fibrous stroma. The cell bodies were rather widely separated by masses of glial fibers, which were very delicate and cobweb-like in texture. In some areas the tumor closely resembled cerebral tissue with occasional ganglionic cells. The growth did not appear to be very malignant, but was definitely of neurocytic origin, a neuroblastoma. (Fig. 3.)

The only postoperative complication was a foot drop on the right; the neurologic consultant thought this due to injuries to the fifth lumbar and the first sacral nerves. The child is well today over four years after the operation. Apparently there is no recurrence and the foot drop has disappeared.
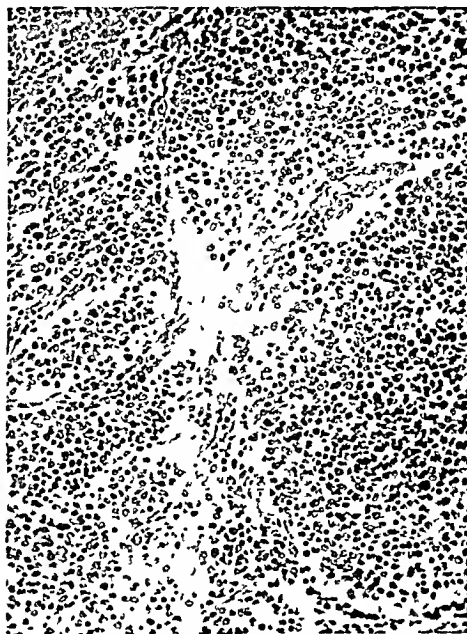

FIG. 2. Case I. Neuroblastoma.

CASE III. This was a female child, aged 26 months. The chief complaint was an abdominal swelling which had been noticed for five weeks and which was growing rapidly.

Examination showed a huge, nodular, firm, and not tender mass which almost filled the abdomen and which was not movable. It was more prominent in the midline, but extended well into both flanks. Roentgen plates showed the bowels displaced by the mass and an enlarged liver. Chest plates showed a marked mediastinal shadow protruding into the left lung. The long bones were negative.

On cystoscopy the P.S.P. function on the right was eight minutes and on the left fourteen. Pyelogram demonstrated marked displacement of both ureters laterally and compression of both ureters. (Fig. 4.)

A transperitoneal exploratory operation was undertaken in spite of the apparent hopelessness of the case. The mass was found to be retroperitoneal and extended from the level of the gastrohepatic omentum down to the bifurcation of the common iliac arteries. The ureters as well as the kidneys were pressed laterally by the mass. There were many enlarged lymph glands in front of the tumor. Since removal was impossible, two of the en-

larged lymph glands were removed for histologic examination and the abdomen was closed.
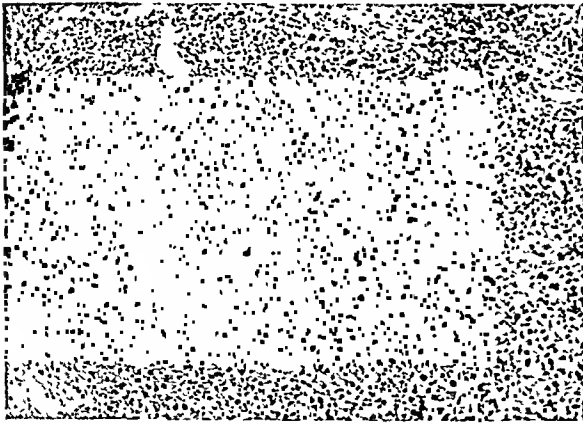
The histologic examination of the lymph

FIG. 3. Case II. Neuroblastoma.

glands showed the nodes replaced by tumor tissue, composed of small cells with deeply stained round vesicular nuclei. The cell bodies were ill defined and consisted of narrow rims of cytoplasm which streamed into fine processes, forming an intercellular glial network. Occasionally the cells were arranged in small rosettes. Numerous mitotic figures were present. The
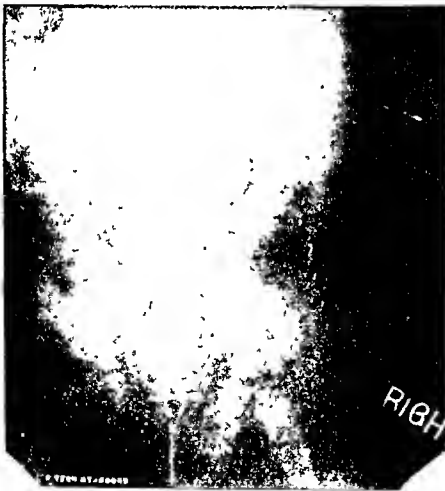

FIG. 4. Case III. Displacement of both ureters.

tumor, a neuroblastoma, was highly vascular and appeared to be very malignant. (Fig. 5.)

The child had an uneventful postoperative course, but the tumor continued to grow and the patient died two months after the exploratory operation.

CASE IV. This woman, 70 years of age, had as chief complaint a swelling in the right side of the abdomen which had been present for eleven months and which caused a great deal of pain. She stated that the mass has been

FIG. 5. Case III. Neuroblastoma.

growing slowly, but that lately the pain had been getting very much worse. Her general condition was poor and she had suffered for many years from hypothyroidism and adiposity.

Examination showed a huge mass in the right side of the abdomen. It was irregular in outline, not movable and quite tender. Distinct fluctuation could be felt. As far as we could tell it extended from the costal margin to somewhere below the iliac crest and seemed to be retroperitoneal; intestines could be palpated in front of the mass.

A barium meal merely showed displacement of the bowel by the mass and a vaginal examination seemed to rule out tumor arising from the pelvic organs.

On cystoscopy a steady drip of hazy urine was observed to come from the right kidney, which continued during observation, namely for 30 minutes. P.S.P. appeared from the left in four minutes, but none appeared from the right during the period of observation. A right pyelogram with 20 c.c. of contrast medium showed a huge misshapen pelvis, but no calices. The outline of the kidney could not be discerned.

Because of the pain and in spite of the patient's poor general condition, operation was undertaken for drainage of the hydronephrotic sac. Under local anesthesia the lumbar ab-

dominal wall was dissected down to the mass and 1,500 c.c. of a light brown watery fluid drained off. At the end of the drainage blood
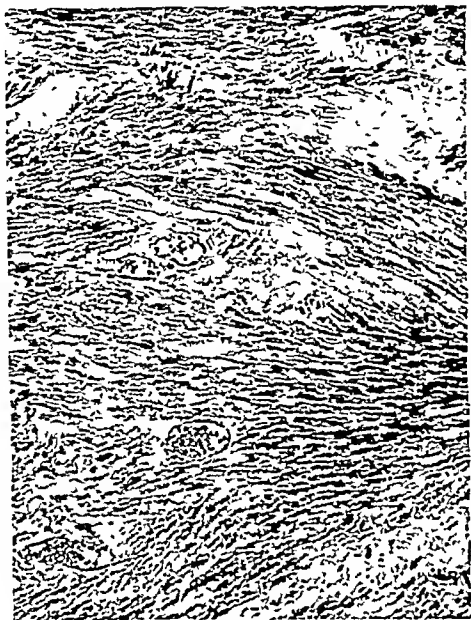

Fig. 6. Case iv. Leiomyosarcoma.

appeared through the trocar. A gauze pack and compression seemed to control the bleeding, but the patient died a few hours later.

The post-mortem examination revealed a very complex state of affairs. The nucleus of the pathologic changes found was a large hard nodular tumor in the right lumbar gutter. It extended from the level of the second lumbar vertebra to the upper margin of the sacrum. Its upper pole was attached to the right kidney and had invaded and compressed the vena cava. The compression of the vena cava had produced numerous huge varices of the veins of the abdominal wall. The fluid drained off during the operation had been in a retroperitoneal, but extrarenal cavity. It is probable that it formed on the basis of an old hematoma. The operative hemorrhage was due to a ruptured varix. The right kidney was totally hydronephrotic, and the right ureter was compressed along its upper abdominal portion by the tumor.

Histologic examination showed large spindle-shaped cells arranged in broad interlacing bundles. The nuclei were generally oval and vesicular, but many of them were very large, hyperchromatic or showing mitotic figures. Occasional multinuclear forms were seen. The cell bodies were large and stained yellow by the

Van Gieson method. A scanty connective tissue stroma distributed throughout the growth carried a fairly abundant vascular supply. The
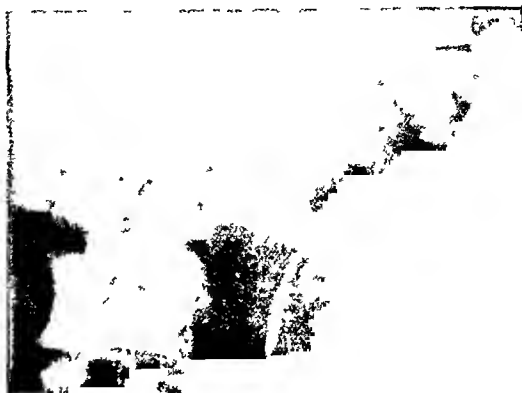

Fig. 7. Case v. Note left hydronephrosis and compression of left ureter.

tissue was rather edematous. Diagnosis was leiomyosarcoma. (Fig. 6.)

CASE v. The patient was a male 39 years of age whose chief complaint was pain in the region of the left kidney of three months' duration. His abdomen was too fat to allow accurate palpation. A pyelogram showed marked
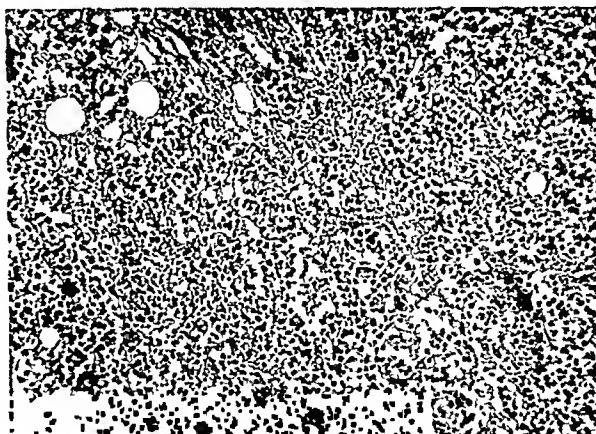
Fig. 8. Case v. Metastatic embryonal carcinoma of testis.

hydronephrosis of the left kidney with decreased function and marked and persistent compression of the left ureter below the kidney pelvis. (Fig. 7.) On operation a hydronephrotic kidney was found. The compression of the ureter was due to an extrarenal tumor which extended from the pedicle of the kidney down to the third lumbar vertebra. The tumor was smooth, white and firm. It was densely attached to the aorta. Removal of the kidney and the tumor was undertaken, but it could not be

separated from the aorta wall. A small strip of tumor tissue had to be left behind, measuring about 7 cm. in length.

The tumor was composed of broad sheets and nests of rounded or polyhedral cells, separated by a delicate stroma network and occasional broader trabecular bands. The tumor cells were of fairly uniform size and shape. Their nuclei were rounded and vesicular; the cell bodies were pale and vacuolated. In the stroma were abundant lymphocytes. (Fig. 8.) The diagnosis was metastatic tumor probably arising from an embryonal carcinoma of the testicle.

Examination of the testis showed no increase in size and no nodules, but the left epididymis was distinctly thickened and indurated. Nevertheless, it was decided to remove the testicle. Even after the testis was taken out of the scrotum it was impossible to discern any tumor, and only after removal and cross-section, was a pea sized, yellow tumor found in the center of the testicle. Histologic sections of this tumor showed marked central necrosis, but the marginal portions showed the same histologic picture as the metastatic tumor.

The patient made an uneventful recovery, and was then treated with deep Roentgen rays. He is alive and apparently well two years after the operation.

SUMMARY

Three cases of retroperitoneal neuroblastomata in children are reported. Apparently they differ widely in their degree of malignancy. Removal of such tumors should be attempted since cure has been effected in two of the three cases reported here. The second case is particularly remarkable, because removal of the tumor seemed to be incomplete and yet the child made a complete recovery.

One case of retroperitoneal leimyosarcoma is reported which is remarkable on account of the complexity of the pathologic findings.

One case of metastatic testicular tumor in the retroperitoneal space is reported. It tends to corroborate the fact that such tumors can be cured by deep roentgen therapy.

Grateful acknowledgment is due to Drs. D. D. Davis and W. H. Field for some of the clinical material reported here and to Dr. J. A. deVeer for the careful analysis of the pathologic findings.

REFERENCES

1. ALYEA, E. P. Medulloblastoma involving kidney. *South. M. J.*, 26: 753, 1933.
2. BARRINGER, B. S. Radiosensitive kidney tumors. *J. Urol.*, 38: 1, 1937.
3. BIGLER, J. A., and HOYNE, A. Ganglioneuroma. *Am. J. Dis. Child.*, 43: 1552, 1932.
4. BOTHE, A. E. Neurocytoma of the kidney. *Ann. Surg.*, 101: 418, 1935.
5. CULVER, H., and DAVISON, M. Tumors of the adrenal gland. *J. Urol.*, 32: 428, 1934.
6. HOFFMAN, S. J. Neurocytoma. *Am. J. Dis. Child.*, 49: 135, 1935.
7. JACKSON, C., and BABCOCK, W. W. Retroperitoneal ganglioneuroma. *S. Clin. North America*, 11: 1233, 1931.
8. JERGENSEN, F. H. Hypertension and retroperitoneal ganglioneuroma. *Arch. Path.*, 16: 340, 1933.
9. LEWIS, D., and GESCHICKTER, C. F. Tumors of the sympathetic nervous system. *Arch. Surg.*, 28: 16, 1934.
10. McFARLAND, J. Ganglioneuroma. *Arch. Path.*, 11: 118, 1931.
11. MORGAN, E. K., and STONE, C. M. Primary retroperitoneal leiomyosarcoma. *J. Urol.*, 39: 63, 1938.
12. WEHRBEIN, H. L. Ganglioneuroma of the retroperitoneal space. *Long Island M. J.*, vol. 23, 1929.

DIDELPHYS IN SISTERS

G. T. Tyler, Jr., m.d.

GREENVILLE, SOUTH CAROLINA

E. T., white, age 12, was admitted to the Greenville General Hospital in September, 1937, complaining of vaginal bleeding. Her periods began at 11 years of age. She menstruated regularly for four months, missed two months, then began bleeding, and continued to bleed until admission. She was well developed, apparently 16 years old. Except for pallor and tenderness in the lower half of the abdomen, the general physical examination was negative. A possible pregnancy was excluded by a negative Friedman test. On examination under anesthesia, there was no abnormality of cervix or vagina. A uterine sound introduced through the cervix, passed to the left. It could not be directed to the right. There was a mass about 4 cm. in diameter, round, to the right, and close to the body of the uterus, which was inclined to the left. Double uterus and ovarian cyst were considered as possible diagnoses. Laparotomy at a later date was decided upon.

When the abdomen was opened, the pelvic structures appeared as in Figure 1. Removal of the mass to the right of the uterus was attempted. This had partly succeeded when we encountered bleeding which we could not control. It was decided to remove the uterus supravaginally to check the bleeding. This was done, but it was necessary also to pack the wound before bleeding ceased. The left ovary was retained. The wound was closed around the packing.

The patient lost a great deal of blood. Hard work by the internes and immediate transfusion, repeated the following day, saved her life. She finally recovered, and left the hospital with the wound nearly healed. When seen three months later, she was in good health. It was explained to the mother that the child could not menstruate and could never bear children.

The pathologist reported that there were two bodies to the uterus, one tube attachment to each. Several pieces of tissue were removed from a cyst wall and section showed a denuded endometrium. Microscopically, the cyst wall had fibromuscular stroma, covered by stratified squamous epithelium. This was denuded, and had granulation tissue. The cyst contained simple columnar epithelium. The tissue suggested congenital misplaced vaginal structure.

The left cornu had been curetted shortly before operation. The "denuded" areas were in this curetted cornu.

Four months later, in January, 1938, the patient's sister, A. T., aged 14, was admitted. She complained of pain in the lower abdomen for the past three months and cessation of periods. She had not been so uncomfortable since periods ceased as she was while they had been present. Menstruation had begun at 11 years of age. With onset of periods, there was nausea, vomiting, and confinement to bed, often for a week.

This patient also was very well developed for her age. There was nothing unusual in the general physical examination.

Under anesthesia, the vagina and the cervix were examined and found normal. A uterine sound passed to the left into the uterus. A mass slightly larger than the uterus lay closely adherent to the right side. The sound could not be introduced into this mass. We made a diagnosis of double uterus, and immediately opened the abdomen. (Fig. 2.)

The right cornu was larger and softer than the left and was removed with its tube. No communication could be found between its cavity and the cervix or vagina. The epithelium remaining was curetted and cauterized with iodine. The raw surfaces were peritonized, the appendix removed, and the abdomen closed. Recovery was uneventful. The cornu of the uterus was hypertrophied, and showed a thickened, softened, and congested endometrium in longitudinal folds, as if it had been distended.

In September, 1938 both patients were in good condition.

It is unusual for double uterus to occur in sisters. An interesting feature of the history is that their mother is the second wife, and that among four children from the father's first marriage there was no complaint of "female trouble." A visual and bimanual pelvic examination of these pa-

tients' mother showed normal vagina and cervix. The abdominal wall was so thick that nothing could be made out bimanually.
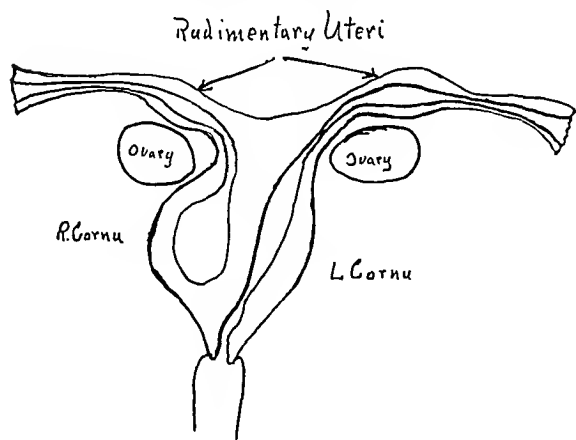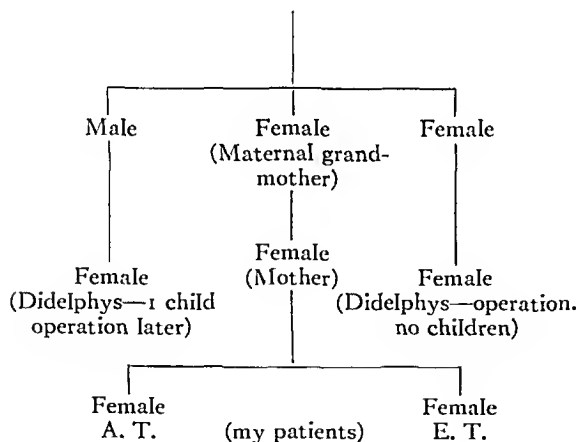

FIG. 1. Case I.

An interesting family history was later obtained from the mother who reported that two maternal cousins had double uterus, proved by operation. One of these cousins had borne a child before she was operated upon.

Conservative treatment of didelphys is generally agreed upon among gynecologists. Many such patients have had normal pregnancies. Some patients have had a normal pregnancy in one horn and a later pregnancy in the other horn. Some cases have been discovered only at operation, the previous history giving no hint of unusual conditions. Nearly 50 per cent of preg-

nancies occurring in such patients terminate in miscarriage.
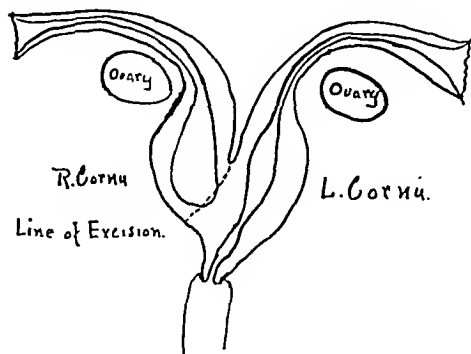
In the first case, had we been fortunate

FIG. 2. Case II.

enough to remove the right cornu, it is interesting to speculate on the possibilities of pregnancy and its outcome. The small masses at the uterine ends of the tubes were undoubtedly rudimentary uteri, also supernumerary. Fertilization having occurred in the tube, the ovum would most likely have lodged in this structure. If it matured, Cesarean section would probably have been necessary.

In the second case, one apparently normal horn of the uterus remains with its tube and two ovaries. The possibility of pregnancy is scarcely less than in a normal uterus one of whose tubes has been removed.

SUMMARY

An instance of didelphys in sisters is presented. Probably other such cases are known but have not been published. Frequently the condition is unrecognized. Discovery is not an indication for operation unless symptoms demand it. If operation is done, the surgeon should be as conservative as possible. A secondary more radical operation is preferable to depriving the patient of her child-bearing function unless this is clearly indicated. It is better to allow the patient a test of pregnancy. She or her family should be told of the condition; and if pregnancy occurs, she should be kept under close observation.

MALIGNANCY OF THE ANUS, RECTUM AND SIGMOID COLON IN THE YOUNG*

WITH REPORT OF A CASE AT FOUR AND A HALF YEARS

HARRY E. BACON, M.D., F.A.C.S., F.A.P.S.   AND   W. BURGESS SEALY, M.D.

Assistant Professor of Proctology, Temple University School of Medicine and University of Pennsylvania Graduate School of Medicine

Fellow in Surgery, Mayo Clinic

PHILADELPHIA, PENNSYLVANIA   ·   ROCHESTER, MINNESOTA

UNTIL the etiology of cancer is known and a specific treatment developed; while our research colleagues continue their own experiments and evaluate the numerous hypotheses and therapies suggested by others; the medical profession at large must continue the education of the laity in regard to malignancy, and we, the clinicians, must content ourselves with endeavoring to put into as many hands as possible the only weapon at present at our command—early diagnosis.

Carcinoma of the rectum and sigmoid colon in adults is rather common, comprising approximately 12 per cent[104] of all malignant neoplasms. This figure is sufficiently high to arouse every physician to suspect a malignant process when a history of bleeding or change in bowel habit is given by the patient. In youth, however, the occurrence of malignancy is computed at 4 per cent,[103] and of this only a small proportion can be assigned to the lower bowel. Nevertheless, the fact that even a relatively small number of tumors have been and continue to be found in the rectum and sigmoid in children should prompt us to suspect malignancy at any age, even though the clinical train of symptoms is obscure or indefinite.

Fowler,[31] in an excellent article on malignancy in persons under 26 years of age, noted that 18.7 per cent of neoplasms were in the large bowel, and of these 12.5 per cent were located in the rectum and rectosigmoid. A review of several large series shows that cancer of the distal bowel is infrequent in youth and early adult life. (Table I.)

TABLE I

Investigator	Number of Cases	Percentage below 30 Years	Percentage below 20 Years
Matzen[67]	8054	......	.003
Pennington[82]	7174	.032	.005
Rosenberg[95]	4048	......	.002
Bacon[5]	1995	5.4	.004
Rankin[92]	1452	3.85	
Janusz[48]	831	......	.0024
Wildholz[114]	563	1.95	
Glazer[35]	527	3.5	
Lynch[60]	491	7.0	
Passler[80]	282	1.0	
Schreiner[101]	210	2.6	
Brindley[11]	100	2.0	
Fitzwilliams[29]	....	11.0	4.0

In spite of such evidence, however, we should be ever mindful of the possibility of such conditions in youth. Just recently a case came under our own observation, which is reported here.

CASE REPORT

H. L., a lad of 4 years, 7 months, was brought to the Children's Clinic of the Philadelphia General Hospital because of bleeding and protrusion from the bowel. As related by the child's mother, about one and a half years before blood was first noticed in the toilet bowl after a bowel movement. During the ensuing four months the stools were streaked with blood about once a week; but for the past year bleeding had

---

FIG. 1. Fungating protruding adenocarcinoma of rectum in a young man of 17 years. (Courtesy of C. F. Martin.)

FIG. 2 Boy 4 years and 7 months old, with early carcinoma of rectum.
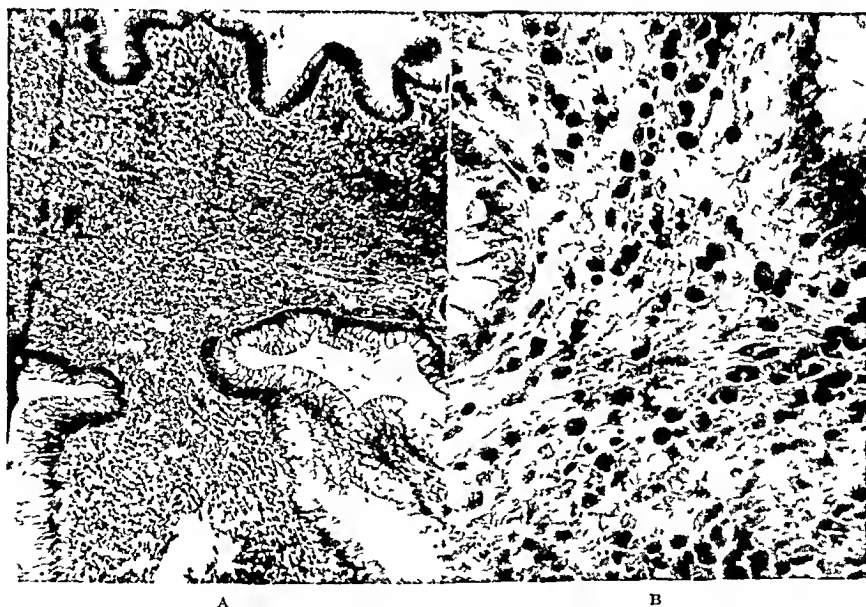
FIG. 3. A, low power section through polypoid growth of rectum. Note hyperplasia of acinar epithelium and inflammatory changes in stroma. B, high power magnification of same specimen, showing poorly defined basement membrane. Some of glandular components are lined by columnar goblet cells, presenting normal maintenance of nuclear polarity, while a few exhibit hyperchromatic cell lining accentuated by definite loss of nuclear polarity assuming pseudostratified and stratified arrangement, mitotic figures being frequently encountered.

been more or less constant following each defecation. About four weeks previous, approximately a teaspoonful of fresh blood was passed, and this situation had persisted until the time of admission to the Clinic.

When blood was first noticed, the little patient was examined by his parents, who found a blood-streaked mass about the size of the rubber tip of an ordinary lead pencil protruding from the anus. The small mass was pushed back into the rectum with a finger covered with toilet paper and vaselin. No pain had been experienced by the child at any time.

In his history, mumps, measles, chicken pox, and whooping cough were cited. Both the father and mother were living and well, aged 32 and 29 respectively. No family history of cancer was obtained.

The patient was referred to the Proctoscopic Clinic, where a polypoid growth about the size of the distal phalanx of the index finger was found. Dr. Philip R. Custer, pathologist of the Philadelphia General Hospital reported that this was a firm, encapsulated mass, 15 by 12 by 8 mm., accompanied by a small piece of firm, pale gray tissue. On cross section the mass appeared to be composed of mottled gray and purple tissue.

The surface of the polyp was denuded of epithelium and underlaid by a zone of acute and chronic inflammation. Some of the glandular components were lined by columnar goblet cells, showing normal maintenance of nuclear polarity. A few, however, exhibited hyperchromatic cell lining accentuated by definite loss of nuclear polarity, assuming pseudostratified and stratified arrangement. Mitotic figures were frequently encountered. Dr. Custer was inclined to regard these as evidence of early malignancy. Two other pathologists have confirmed this diagnosis.

The patient was admitted to the children's department June 21, 1937 and a radiologic consultation was requested. Digital and proctosigmoidoscopic examination elicited no pathology; yet, because the child was obstreperous, reëxamination under vinethene was considered expedient. This was also negative.

Dr. B. Holmes three days later reported that fluoroscopic examination of the rectum and sigmoid showed no pathologic lesion. The large intestine was apparently normal. A contrast enema showed the pelvic, transverse and ascending colon well filled with air, barium

being present only in the rectum, a part of the sigmoid and cecum. There was no evidence of polyposis. This study was repeated on June 28 and the findings essentially confirmed.

The urine examination was negative. Blood count showed: erythrocytes, 4,180,000; leucocytes, 7,700; hemoglobin, 78 per cent; differential: polymorphonuclear leucocytes, 50 per cent; lymphocytes, 48 per cent; transitionals, 2 per cent. Blood sugar was 87 mg. per 100 c.c.; blood urea, 14 mg. per 100 c.c. The Kahn test was negative.

The patient was discharged from the hospital on June 30, 1937 but has been followed in the Radiologic Clinic periodically. There has been no evidence of recurrence, and the child has gained weight.

A review of the literature citing proved cases of anal, rectal, and sigmoidal malignancy in patients 19 or under has been condensed into Chart 1.

Warthim[112] has mentioned that malignant tumors are relatively rare before puberty, but that there is a steadily ascending line of occurrence from childhood on to middle age, both sarcomata and carcinomata appearing with about equal frequency. In this series of collected cases it will be noted that the largest number for any age below 12 is four, but that above 12 the smallest number of cases for any age is at least twice that number. It is also to be noted that there is an increasing number with each ascending age group. This table shows that there are sixty-five males and twenty-eight females, a ratio of 2.32 to 1. The site was in the rectum in eighty-five cases, the sigmoid in nineteen, the anus in one, and unstated in sixteen.

The symptoms as recorded in thirty-five cases are given in Table II. This is in contrast to the symptoms in adults as reported by one of us.[4] (Table III.)

TABLE II

Abdominal pain	17
Bleeding	9
Constipation	7
Rectal pain	4
Diarrhea	3
Lumbar pain	1
Loss of weight	1

CHART I

	Age	Sex	Location	Symptoms	Duration of Symptoms	Duration of Life	Type of Tumor
Ahfeld[1]	Newborn monster	.	Rectum				
Clar[15]	3	M	Sigmoid				
Lasnier[54]	3		Rectum				Epithelioma
Ullhorn[111]	3½	M	Sigmoid				
Goodman[37]	4	F	Sigmoid	Chronic constipation	4 weeks		Lymphosarcoma
Duncan[28]	5	M	Rectum				
Ullhorn[111]	5	M	Rectum				
Lockhart-Mummery[58]	8		Rectum				
Philipp[84]	9	M					
Steiner[109]	9	M	Sigmoid	Pain in abdomen	2 weeks		
Leijer[57]	9	M	Sigmoid	Constipation	11 days		
Parkinson[79]	9	M	Rectum		6 months		
Rowntree[93]	10	M	Rectum				Sarcoma
Stern[110]	11	F	Rectum	Abdominal pain			
Smith[105]	11	M	Rectum	Bleeding	6 months		Adenocarcinoma
Bethe[8]	11		Rectum				
Rankin[91]	11		Rectum				Lymphosarcoma
May[68]	12	F	Rectum				
Paultauf[81]	12	F	Sigmoid				
Garrard[34]	12	M	Sigmoid	Constipation			
Zuppinger[116]	12	F	Sigmoid	Abdominal pain	3 months		
Parkinson[78]	12	F	Rectum	Abdominal pain			Adenocarcinoma
Marchand[63]	12	F	Anus	Rectal pain	9 months		
Cummings[21]	12	M	Rectum				
Busche[12]	12	M	Rectum	Pain in rectum			
Mayo[69]	12	M	Rectum				
Philipp[84]	12	F					
Milne[72]	12	M	Rectum	Intestinal obstruction	Few days		Colloid
Mandl[62]	12		Rectum				
Nothnagel[74]	12	F	Sigmoid				
Jordan and Chamberlain[49]	13	M	Rectum	Diarrhea	21 months		Adenocarcinoma
Czerny[22]	13	M	Rectum		18 months		Adenocarcinoma
Allingham[2]	13	M	Rectum				
Gurlt[41]	13	M	Rectum				
Gowland[38]	13	M	Rectum				
Kupfelere[52]	13	F	Rectum				
Raiford and Buttles[89]	13	F	Rectum	Lumbar pain	4 weeks		Colloid carcinoma
Babcock[3]	13	F	Rectum				
DeSaive[25]	13		Rectum				
Bottin[10]	13	F	Rectum				
Rostoker[97]	14		Rectum				
Schneider[99]	14						
Philipp[84]	14	M					
Mayo-Robson[70]	14	.	Rectum				
Caird[13]	14						
Davis[24]	14	M					Adenocarcinoma
Mandelung[61]	14		Sigmoid				
Olmsted[76]	14	M	Sigmoid	Abdominal pain and diarrhea	4 months		Adenocarcinoma
Porter[87] .	14						
Cripps[19]	14	M	Rectum		2 months	3 months	Adenocarcinoma

CHART I (Continued)

	Age	Sex	Location	Symptoms	Duration of Symptoms	Duration of Life	Type of Tumor
Israel[46].	14	M					
Michauz[71]	15	M	Rectum				Colloid
Wilde[113]	15	M	Rectum				
Marsh[64] .	15	M	Rectum	Abdominal pain	6 months		Adenocarcinoma
Bernouillc[7]	15	M	Rectum				
Clogg[17]	15	..	Rectum				
Porter[87]	15						
Godin[36].	15	.	Sigmoid				Scirrhous carcinoma
Yeomans[115]	15	M	Rectum				
Gant[33]	15	F	Rectum				
Fowler, R II.[32]	15	M	Rectum	Pain in rectum and abdomen	9 months		Adenocarcinoma
Philipp[84]. . .	15	M					
Rome[94] ...	15	M	Rectum	Abdominal pain	1 month		Adenocarcinoma
Schneider[99]	15						
Rostoker[97]	15						
Kaleniewicz[50]	16	M	Sigmoid				
de la Camp[53]	16	.	Rectum				
de la Camp[53]	16		Rectum				
Gant[33]	16	M	Rectum				
Clark[16]	16	M	Sigmoid	Abdominal pain	6 months	6 months three days	Adenocarcinoma
Pillon[85]	16	F	Rectum	Abdominal pain	3 months		
Grulee[40]	16	F	Sigmoid	Abdominal pain			Adenocarcinoma
azarus-Barlow[56]	16	M	Rectum				Colloid
Porter[87]	16						
Martin, C. F.[65]	16	M	Rectum	Bleeding	8 months	15 months	
Rankin and Chumley[90]	16	.	..				Colloid
Steffins and Burkc[108]	17	M	Rectum				Colloid
Orr[77]	17	M	Rectum				
Gant[33]	17	M	Rectum				
Gant[33]	17	F	Rectum				
Lawson[55]	17	M	Rectum	Bleeding	7 months	7½ months	Colloid
Senn[102]	17	M	Rectum				
Colwell[18]	17	M	Rectum				
Oehler[75]	17						
Allingham[2]	17	M	Rectum				Encephaloid
Foges[30]	17	M	Rectum		8 months		
Schoenning[100]	17	F	Rectum	Pain in rectum, bleeding			
Schoenning[100]	17	F	Rectum	Abdominal pain and constipation			
Mathews[66]	17	F	Rectum		2 years	2 years	
Porter[87]	17	.	Rectum				
Quenu[83]	17	F	Rectum				
Hayden and Shedden[42]	18	F	Rectum				
Gant[33]	18	F	Rectum				
Steffins and Burkc[108]	18	M	Sigmoid				
Gant[33] .	18	F	Rectum				Colloid
Lowenberg[59]	18	M	Rectum	Diarrhea	6 weeks	..	Adenocarcinoma
Spittler[107]	18	M	Rectum	Constipation bleeding	3 months	6 months after operation, 10 months after first symptom	Colloid
Billroth[9]	18		Rectum				

CHART I (*Continued*)

	Age	Sex	Location	Symptoms	Duration of Symptoms	Duration of Life	Type of Tumor
Heuck[45]	18	M	Rectum	Constipation, pain in abdomen	17 months		
Hayd[44]	18	M	Rectum	Abdominal pain, bleeding	18 months		Scirrhous
Miles	18	..	Rectum	. . ..	. . .. .		Carcinoma
Hastings[43]	18	M	Rectum		4 months	14 months	
Csesch[20]	18	M	Rectum	Bleeding	8 months	....	Adenocarcinoma
Cripps[19]	18	M	Rectum	..	4 months	10 months	
Carnett[14]	18	M	Rectum				
Steffins and Burke[103]	19						
Smith[106]	19	M	Rectum		.. .	2 years after colostomy	Adenocarcinoma
Ross[96]	19	M	Rectum	Abdominal pain	2 weeks	4 weeks after onset	
DaCosta[23]	19	M	Rectum	Constipation	4 months		Adenocarcinoma
Gant[33]	19	F	Rectum				
Molliere[73]	19	F	Rectum				
Miles	19	.	Rectum	.	. . ..	.. ..	Carcinoma
Billroth[9]	19		Rectum				
Dunbar[27]	19	M	Sigmoid	Abdominal pain	2 months		Colloid
Gurlt[41]	19	.	Rectum				
Phifer[83]	19	M	Sigmoid	Loss of weight and diarrhea	6 months		
Barber[6]	19	M	Sigmoid	Intestinal cramps	1 year		Adenocarcinoma
Rohde[93]	19	M	Rectum	Bleeding			
Platt[86]	19	M	Rectum		8 months	Living at end of one year	
Jacobs[86]	19	F	Rectum				
Dieterich, H.[26]	19	F	Rectum			.	Adenocarcinoma
Grave[39]	19	M	Rectum				
Kiger[51]	19	F	Rectum				

By comparison of these tables it will be noted that abdominal pain was the most frequent symptom in youth, whereas bleeding was the most common in adults.

TABLE III

	No.	Per Cent
Bleeding.	574	31.5
Change in bowel habit .		
Constipation .	289	15.8
Diarrhea.	269	14.7
Frequent or urgent desire for stool.	195	10.7
Pain:		
Discomfort (local)	101	
As pain or tenesmus	391	
Total	1819	

The histologic type of tumor was reported in only thirty-seven instances. (Table IV.)

TABLE IV

Adenocarcinoma	19
Colloid	10
Scirrhous	2
Encephaloid	1
Gelatinous	1
Epithelioma	1
Lymphosarcoma	2
Sarcoma	1

As Rankin has said, the active tissues of youth, instead of resisting invasion of carcinoma, invite its spread, and the young person who is host to malignant neoplasm has little chance for longevity, regardless of the type of therapeutic measures instituted.

Shedden,[103] in a report of thirty-six collected cases, states that in twenty-five

operation was done, while eleven were untreated. Of these, all except three of those operated upon were dead at the end of twenty-four months, and twenty-five of the original thirty-six were dead within nine months. In our collected series the duration of symptoms at the time of diagnosis, as estimated in thirty-four cases, was 6.9 months. The average duration of life as mentioned in eight cases was ten months and three days.

SUMMARY

1. A case of early carcinoma of the rectum in a male 4 years and 7 months of age is reported.

2. The literature has been reviewed, citing 123 authentic cases of primary malignancy of the anus, rectum or sigmoid colon below the age of 20.

CONCLUSIONS

1. Any patient, independent of age, who complains of pain in the abdomen, rectum or lumbar region deserves a complete study of the lower bowel, employing digital examination, proctosigmoidoscopy, and roentgenography.

2. There is no true cancer age.

3. All tissues removed should be examined microscopically as a routine procedure.

4. Thorough periodic examinations should be made for recurrence, metastasis, and other neoplasms.

REFERENCES

1. AHFELD, F. Zwei Casuistik der congenitalen Neoplasm. *Arch. f. Gynäk.*, 16: 141, 1880.
2. ALLINGHAM, W., and H. W. Diagnosis and Treatment of Diseases of the Rectum, 7th ed., p. 328. New York, 1901. Wm. Wood & Co.
3. BABCOCK, W. W. Personal communication.
4. BACON, H. E. The Anus, Rectum and Sigmoid Colon. Philadelphia, 1938. J. B. Lippincott Co.
5. BACON, H. E. Résumé of experimental studies in proctology. *J. Med. World*, Nov. 1934.
6. BARBER, W. H. Intestinal obstruction caused by adenocarcinoma of the sigmoid in a boy 19. *Interstate M. J.*, 3: 106, 1919.
7. BERNOUILLES, E. Magen—Darmskrebs in den beiden ersten Lebensezennien. Inaug. Diss. Berlin, 1907.
8. BETHE. Quoted by Pennington. Rectum, Anus and Pelvic Colon, p. 764. Philadelphia, 1923. P. Blakiston.
9. BILLROTH, H. *Th. Chir. klin. Berlin*, 290, 1878.
10. BOTTIN, J. *Ann. Soc. chir. de Liège*, 168: 174, 1935.
11. BRINDLEY, G. V. Cancer of the rectum. *South. M. J.*, 25: 44 (May) 1932.
12. BUSHE, G. Treatise on Malformations, Injuries and Diseases of Rectum and Anus, p. 292. New York, 1837. French and Adlard.
13. CAIRD, F. M. *Liverpool M. J.*, 293 (July) 1913.
14. CARNETT, J. B. Quoted by J. C. Howell.
15. CLAR. Quoted by Nothnagel.[74]
16. CLARK, J. H. Cancer of the sigmoid and rectum in children and young adults. *Ann. Surg.*, 84: 833, 1926.
17. CLOGG, H. S. Cancer of the colon, a study of 72 cases. *Lancet*, 2: 1007 (Oct. 3) 1908.
18. COLWELL, H. *Arch. Middlesex Hosp.*, 6: 141, 1905.
19. CRIPPS, H. Cancer of the Rectum, 5th ed., p. 186. London, 1907. J. & A. Churchill.
20. CSESCH, P. Beitrag zur statistik der Rektumcarcinoma. Inaug. Diss. Breslau, 1897.
21. CUMMINGS, J. P. Malignant disease of the rectum in a boy 12. *Am. J. M. Sc.*, 53: 353, 1854.
22. CZERNY, V. Mastdarmkrebs bei einem 13 jahr Knaben. *Munchen. med. Wchnschr.*, 43: 241 (Mar.) 1896.
23. DACOSTA, J. C. Carcinoma of the rectum. *S. Clin. North America*, 8: 69, 1928.
24. DAVIS, A. S. Adenocarcinoma in a fourteen year old boy. *Calif. & West. Med.*, 31: 139 (Aug.) 1929.
25. DESAIVE, P. Un cas rare d'adenocarcinome anorectal. *Liège Méd.*, 27: 945 (July 22) 1934.
26. DIETERICH, H. Radical treatment of cancer of the rectum from the experience of the Giessen Clinic. *Arch. f. klin. chir.*, 150: 691, 1928.
27. DUNBAR, L. Carcinome colloide de l'iliaque chez un jeune homme de 19 ans. *Bull. et mém. Soc. anat. de Paris*, 54: 644, 1879.
28. DUNCAN. Quoted by Walker and Daly. *Oklahoma M. J.*, 27: 119 (April) 1934.
29. FITZWILLIAMS, D. C. L. Carcinoma of the rectum. *Lancet*, 226: 842 (April 21) 1934.
30. FOGES, A. *Wien. med. Wchnschr.*, 2152 (Oct.) 1913.
31. FOWLER, L. H. Malignant epithelial neoplasms, carcinoma and epithelioma occurring in persons under 26 years of age. *Surg., Gynec. & Obst.*, 43: 73, 1926.
32. FOWLER, R. H. Carcinoma in the young. *M. Rec.*, 87: 730, 1915.
33. GANT, S. W. Diseases of the Anus, Rectum and Colon, Vol. II, p. 298. Philadelphia, 1923. W. B. Saunders Co.
34. GARRARD, W. A. A case of carcinoma of the colon occurring in a child. *Quart. J. Med.*, 5: 234, 1896.
35. GLASER. Quoted by Lynch.[60]
36. GODIN. Quoted by Molliere.[73]
37. GOODMAN, A. L. Lymphosarcoma of sigmoid *Arch. Pediat.*, 33: 771 (Oct.) 1916.
38. GOWLAND. Quoted by Allingham.[2]
39. GROVE, L. W. Carcinoma of the rectum in a boy 19 years of age. *South. M. J.*, 21: 309, 1928.
40. GRULEE, C. G. Carcinoma in early life with a report of a carcinoma of the rectum in a girl 16 year old. *Surg., Gynec. & Obst.*, 2: 678, 1906.
41. GURLT, E. Beitrage zur chirurgischen Statistik. *Arch. f. klin. Chir.*, 25: 421, 1880.

42. HAYDEN, E. P., and SHEDDEN, W. M. Carcinoma of the rectum. *Surg., Gynec. & Obst.*, 51: 783 (Dec.) 1930.

43. HASTINGS. Quoted by Cripps.[19]

44. HAYD, H. Scirrhous cancer of the rectum in a boy 18 years old. *Buffalo M. & S. J.*, 22: 497, 1883.

45. HEUCK, G. Zur Statistik und operative Behandlung der Mastdarmkrebs. *Verband. d. deutsch. Geselsch. f. Chir.*, 12: 183, 1883.

46. ISRAEL. Quoted by Phillips.[84]

47. JACOBS, A. W. Carcinoma of the rectum and sigmoid. *Surg., Gynec. & Obst.*, 43: 50 (July) 1926.

48. JANUSZ, W. Cancer statistics on necropsy material of pathologic institute of University of Lwow. *Ztschr. f. Krebsforsch.*, 23: 47, 1926.

49. JORDAN, S., and CHAMBERLIN, D. T. Cancer of the digestive tract in the young. *S. Clin. North America*, 17: 815 (June) 1937.

50. KALENIEWICZ, S. Case of carcinoma of the sigmoid in a 16 year old boy. *Polska. gaz. lek.*, 8: 295, 1929.

51. KIGER, W. H. Personal communication.

52. KUPFELERE. Quoted by Rankin, Bargen and Buie, p. 509. Philadelphia, 1932. W. B. Saunders.

53. LA CAMP. Carcinoma in den ersten beiden Lebensdecennien. *Zentralbl. f. allg. Path. u. path. Anat.*, 8: 708, 1897.

54. LASNIER, E. P. Epithelioma of rectum in a child of thirteen. *Arch. Latino-Am. de Pediat.*, 2: 321, 1923.

55. LAWSON, T. C. Carcinoma of rectum in a young man 17 years. *Calif. & West. Med.*, 40: 171 (Mar.) 1934.

56. LAZARUS-BARLOW, W. S. Cancer ages, a statistical study based on the cancer records of the Middlesex Hospital. *Arch. Middlesex Hosp.*, 5: 30, 1905.

57. LEIJER. Enterotomi ved cancer recti. *Hygea*, 39: 138, 1872.

58. LOCKHART-MUMMERY, J. P. Diseases of the Rectum and Colon, 2nd ed., p. 302 Baltimore, 1934. Wm. Wood & Co.

59. LOWENBERG, S. A. Carcinoma in a young man aged 18 years. *M. J. & Rec.*, 127: 183 (Feb. 15) 1928.

60. LYNCH, J. M., and FELSEN, J. Tumors of the Colon and Rectum, p. 97. New York, 1925. Paul B. Hoeber.

61. MADELUNG, O. Anatomisches und chirurgisches über die Flexura coli sinistra. *Arch. f. klin. Chir.*, 81: 206, 1906.

62. MANDL, F. *Deutsch. Ztschr. f. Chir.*, 145 (Feb.) 1922.

63. MARCHAND. Etude sur l'extirpation de l'extrémité inférieure du rectum. Paris, 1873.

64. MARSH, F. Carcinoma of the sigmoid flexure in a boy aged 15 years. *Lancet*, 1: 379, 1902.

65. MARTIN, C. F. Unpublished case record.

66. MATTHEWS. Quoted by Tuttle, J. P. Diseases of the Anus, Rectum and Colon. New York, 1903. D. Appleton & Co.

67. MATZEN, N. Krebstatistik in Boyern und die Verwertung der Ergebnisse zur Frage Entstehung des Krebes *Ztschr. f. Krebsforsch.*, 25: 105, 1927.

68. MAY. Quoted by Tuttle. Diseases of the Anus, Rectum and Pelvic Colon, p. 764. New York, 1903. D. Appleton & Co.

69. MAYO, H. Observations on Injuries and Diseases of the Rectum, p. 52. Washington, D. C., 1853. D. Green.

70. MAYO-ROBSON. Quoted by Lockhart-Mummery. Diseases of the Rectum and Colon, p. 694. New York, 1923. Wm. Wood & Co.

71. MICHAUX, J. *Tr. Acad. Med., Richmond, Va.*, 1890.

72. MILNEX, J. A. Colloid carcinomata at 12 years of age. *Brit. M. J.*, 2: 925 (Oct. 14) 1905.

73. MOLLIÈRE, D. Traité des maladies du rectum et de l'anus, p. 565. Paris, 1877.

74. NOTHNAGEL, H. Diseases of the Intestines and Peritoneum, p. 402. Philadelphia, 1904. W. B. Saunders.

75. OEHLER, J. Rektumcarcinome. *Beitr. z. klin. Chir.*, 88: 593, 1913.

76. OLMSTED, I. Carcinoma of the lower part of the sigmoid in a boy 14 years of age. *Tr. Am. Surg. A.*, 39: 24, 1921.

77. ORR, J. W. Multiple malignant neoplasms. *J. Path. & Bact.*, Pt. 1, 33: 287, 1930.

78. PARKINSON, J. P. Colloid cancer in a girl of 12. *Lancet*, 1: 1525, 1903.

79. PARKINSON, J. P. *Proc. Roy. Soc. Med.*, 7: 9, 1913.

80. PÄSSLER, H. W. Das Schicksal von 282 Kranken mit Mastdarmbrebs bei verschiedenen behandlungsverfahren Sonderdruck. *Chirurg*, 4: 105 (Jan. 7) 1935.

81. PAULTAUF, R. Carcinom der flexura Sigmoidea von einem zwölfjahrigen. Mädchen. *Wien. klin. Wchnschr.*, 13: 197, 1900.

82. PENNINGTON, J. R. Carcinoma of the rectum and pelvic colon. *J. A. M. A.*, 79: 1829 (Nov. 25) 1922.

83. PHIFER, C. H. Cancer of the rectum and sigmoid in childhood and adolescence. *Ann. Surg.*, 77: 711 (June) 1923.

84. PHILIPP, P. W. Ueber Krebsbldungen im Kindesalter. *Ztschr. f. Krebsforsch.*, 5: 326, 1907.

85. PILLON, A. Rétrécissement cancéreux du rectum chez unè juene fille de seize ans et demi. *Bull. Soc. anat. de Paris*, 41: 434, 1856.

86. PLATT, J. F. On excision of the rectum for cancer with recovery of 21 cases. *Med. Chron.*, 1: 419, 1894.

87. PORTER, M. F. Cancer of the rectum and lower sigmoid with report of a unique case. *New York M. J.*, 95: 264, 1912.

88. QUENU. Quoted by Bland-Sutton, J. Tumors Innocent and Malignant, 7th ed., p. 373. London, 1922. Cassell & Co.

89. RAIFORD, T. S., and BUTTLES, E. M. Primary mucoid carcinoma of the rectum in a 13 year old girl. *Ann. Surg.*, 97: 903 (June) 1933.

90. RANKIN, F. W., and CHUMLEY, C. L. Colloid carcinoma of the colon and rectum. *Arch. Surg.*, 18: 129 (Jan.) 1929.

91. RANKIN, F. W., and CHUMLEY, C. L. Lymphosarcoma of the colon and rectum. *Minnesota Med.*, 247 (May) 1929.

92. RANKIN, F. W., and COMFORT, M. W. Carcinoma of Rectum in Young Persons. Collected Papers,

Mayo Clinic, 21: 258, W. B. Saunders, Phila., 1929.

93. Rohde, E. Uber Krebs in junglichen Alter. Inaug. Diss. Griefswald, 1904.

94. Rome, R. M. Carcinoma of the rectum in a boy of 15. *Long Island M. J.*, 7: 248, 1913.

95. Rosenberg, H. Carcinom in junglichen Alter. *Ztscbr. f. Krebsforscb.*, 41: 220, 1934.

96. Rose, L. I. Carcinoma of the rectum in youth. *Am. J. Cancer*, 21: 322 (June) 1934.

97. Rostocker. Quoted by Rankin, F. W., Bargen and Buie, p. 509. Philadelphia, 1932. W. B. Saunders.

98. Rowntree, C. *Proc. Roy. Soc. Med.*, 69 (Nov. 23) 1913.

99. Schneider, F. *Beitr. z. klin. Cbir.*, 26: 405, 1911.

100. Schoening, G. Ueber dar Vorkommen des Masldarmskrebs in den bisten beiden Lebensdecennien. *Deutscb. Ztscbr. f. Cbir.*, 22: 36, 1885.

101. Schreiner, B. F., and O'Brien, J. P. A study of 210 cases of carcinoma of the rectum and anal canal. *Am. J. Roentgenol.*, 25: 654 (May) 1931.

102. Senn, N. A plea for the international study of carcinoma. *J. A. M. A.*, 46: 1254 (April 28) 1906.

103. Shedden, W. M. Carcinoma of the rectum in youth. *New England J. Med.*, 209: 528 (July 14) 1933.

104. Smith, D. Cancer of Anus, Rectum and Rectosigmoid. In Cyclopedia of Medicine, Vol. x, p. 1106. Philadelphia, 1934. F. A. Davis & Co.

105. Smith, D. Carcinoma of the rectum in an 11 year old boy. *Tr. Am. Proc. Soc.*, p. 116, 1934.

106. Smith, D. *Calif. & West. Med.*, 40: 174 (June) 1934.

107. Spittler, F. A. Carcinoma of rectum at eighteen years of age. *Ann. Surg.*, 88: 316, 1928.

108. Stebbins, G. G., and Burke, M. Cancer of rectum and colon. *Am. J. Surg.*, 37: 437 (Sept.) 1937.

109. Steiner, J. Areolarkrebs des Dickdarms bei einem Neunjahrigen Knaben. *Jabrb. f. Kinderb.*, 7: 61, 1865.

110. Stern, C. Zur Kentniss maligner Neubildungen im Kindesalter. *Deutscbe med. Wcbnscbr.*, 28: 494, 1892.

111. Ullhorn. *Deutscbe f.*, 193. 72, 1925.

112. Warthim, A. S. The occurrence of malignant neoplasms in the young. *Arcb. Int. Med.*, 15: 444, 1915.

113. Wilde, M. Ueber das Vorkommen des krebs bei jungendlichen Individuen. Inaug. Diss. Kiel, 19, 1892.

114. Wildholz, E. Uber Krebs bei Jungendlichen. *Ztscbr. f. Krebsforscb.*, 33: 681, 1931.

115. Yeomans, F. C. Proctology, p. 497. New York, 1936. D. Appleton & Co.

116. Zuppinger. Der Darmkrebs im Kindesalter. *Wien. klin. Wcbnscbr.*, 13: 389, 1900.

HODGKIN'S TUMOR OF THE ANTERIOR MEDIASTINUM AND ANTERIOR CHEST WALL*

Moses Behrend, M.D.

Attending Surgeon, Jewish and Mt. Sinai Hospitals; Thoracic Surgeon, Philadelphia General Hospital and State Sanatoria

PHILADELPHIA, PENNSYLVANIA

THE treatment of tuberculosis affecting the lymphatic glands of the neck is frequently surgical. The number of patients seen today suffering from this once common disease is few. The differential diagnosis of enlarged cervical glands usually depended on the result of surgical biopsy and not infrequently cases of Hodgkin's disease were discovered. With diminution of the number of cases of tuberculosis of the cervical lymph chains has apparently also come an apparent decrease in the number of cases of Hodgkin's disease affecting the cervical glands. Whether this diminution is more apparent then real and whether there is any connection between the decrease in the number of cases of tuberculous glands of the neck and Hodgkin's disease affecting the same lymphatic channels I am unable to state.

Hodgkin's tumor of the anterior mediastinum and anterior chest wall is an uncommon condition. Muller recently reported a case of single mediastinal tumor due to Hodgkin's disease. According to Pfahler and others this tumor is radiosensitive and should be treated accordingly when the diagnosis has been positively affirmed. When there is doubt, as there was in the patient under discussion, operation is advisable, if for no other purpose than that of obtaining a biopsy. If, however, biopsy is the only objective, a formal operation is probably not necessary, since with the aid of fluoroscopic guidance and an 18 gauge needle, enough of the tumor can be aspirated for microscopic examination. In fact recently, after discussing the subject with Dr. James Ewing, I tried this method successfully on a patient with a tumor of the posterior mediastinum during the course of an exploratory thoracotomy.

Hodgkin's disease has been described in the past as producing either a benign or malignant tumor, having the structure of a granuloma, although the true nature of the disease according to Arons is still undetermined. Because of this uncertainty the tumor should be subjected to x-ray treatment, since, as stated before, tissues affected by Hodgkin's disease are radiosensitive.

Hodgkin's disease manifests itself in various forms; it may start as a universal enlargement of all the glands of the body, or be limited to the glands of the neck alone, or to a single tumor.

CASE REPORT

F. W., age 23, was admitted to the Jewish Hospital September 18, 1936.

A year and a half before admission a small lump was noticed on the upper left chest near the sternum. The tumor had been incised because it was thought to be an abscess of tuberculous origin, most likely a lymph node. X-ray treatment was given and the lump subsided somewhat but not completely. A short time before the patient's admission to the hospital the tumor began to enlarge and several x-ray studies confirmed the diagnosis of a tumor of the chest wall.

Physical examination showed a firm nonmovable mass at the junction of the left clavicle and sternum, blending with these bones and extending several inches downwards. There was a definite area of dulness over the bulging mass, which extended from the clavicle to the fifth rib. There were no palpable nodes in the neck or inguinal regions. The diagnosis at that time was chondroma of the chest wall. There had never been any report of an accident.

* Read before the Laennec Society, March 29, 1938.

The patient had had the usual diseases of childhood, and in addition was born a deaf mute.

Fig. 1. X-ray before operation. Tumor definitely outlined, anterior portion of thoracic wall and extending under the sternum.

A communication from Dr. Pfahler stated that the tumor mass was approximately 10 cm. by 8 cm., occupying the left intraclavicular region; it was firmly attached to the wall of the chest and involved or developed from the anterior portion of the first and second ribs, or from the costal cartilages of these two ribs. There was no tuberculosis of the lungs or mediastinum. The tumor mass was not encapsulated. Partial destruction of the first and second ribs was present. The tumor was suggestive of chondroma or chondrosarcoma, with calcification. (Fig. 1.)

Laboratory Examinations. Blood count: hemoglobin 65 per cent; red cells, 3,650,000; white cells 9,100; polymorphonuclears 68; lymphocytes 31; eosinophiles 1; no abnormal leucocytes. There was slight achromia, anisocytosis, occasional poikilocytosis. The Wassermann was negative. Blood sugar was 115 mg., blood urea 16 mg., culture of the chest fluid sterile.

Operation. Under nitrous oxide and oxygen anesthesia, a semilunar incision was made around the breast including the left pectoral muscle. A cartilaginous inflammatory tissue enveloping the tumor was revealed after the ribs were removed. A hard inflammatory wall was stripped back from under the sternum and adjacent ribs; in doing so a thick creamy pus was obtained. When the sternoclavicular joint was disarticulated, pus exuded from the sternal ends of the first and second ribs. These two ribs were excised as far back as they were diseased. A small nick was made in the pleura, extending to the pleural cavity, which was
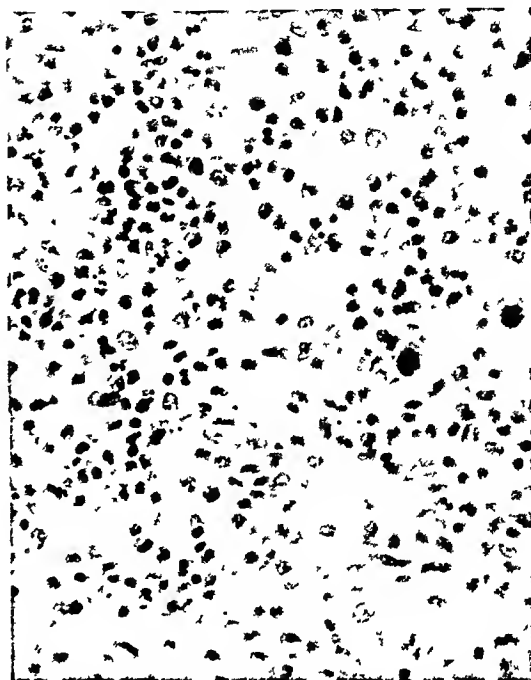

Fig. 2. The pleomorphic nature of the process seen in Hodgkin's disease is exemplified by the presence of lymphocytes, neutrophiles, eosinophiles and large mononuclear cells.

repaired, without subsequent complications. After the tumor and the abscess cavity were dissected away the superficial fascia was closed with catgut. Michel clips approximated the skin.

Pathologic Report (Dr. S. Levine). Specimen consisted of a large number of pieces of tissue, some soft and some bony in consistency. Section presented a large number of inflammatory exudative cells composed of polymorphonuclears, mononuclear cells, lymphocytes and a number of phagocytic cells. There were a large number of fibroblastic and epithelioid cells and also an endothelial proliferation, in addition to reticulum cells. No typical Dorothy Reed cells were noted. However, a number of large endothelial cells with hyperchromatic nuclei were present. Large areas of necrosis were seen throughout, and a considerable amount of fibrosis was observed. There was no definite evidence of tuberculosis. A number of eosinophiles were present.

The diagnosis was Hodgkin's granulomatous tissue undergoing acute inflammatory changes. (Fig. 2.)

In an x-ray report September 30, 1936, Dr. Leon Solis Cohen stated that both diaphragms were normally arched. The cardiac

SUMMARY

A case of Hodgkin's tumor of the anterior mediastinum and chest wall is presented.

FIG. 3. Twelve days after operation. Diffuse haziness over anterior portion of thorax. Definite outline of tumor absent.

FIG. 4. Seventeen months after operation. Entire absence of mediastinal tumor. Portions of first and second ribs absent due to operation.

silhouette was normal in size, shape and position. The mediastinum showed no widening. The anterior portions of the left first and second ribs had been excised. The right lung was normal, while the left showed a diffuse haziness, most likely due to thickening of the parietal pleura or its involvement by the granulomatous mass. There was no definite evidence of a pneumothorax on the left side, but the lung was hypoventilated, probably due to fibrosis. There was no evidence of mediastinal Hodgkin's. (Fig. 3.)

The patient received many x-ray treatments and on February 23, 1938 the heart shadow was reported as normal, the diaphragmatic shadows clear, and the lung fields and ribs as perfectly normal in appearance. No evidence of any mediastinal enlargement nor any Hodgkin's infiltration of any of the ribs was visible. The left first and second ribs were short, due to the former resection. (Fig. 4.)

The condition is extremely rare. The diagnosis may be established by needle biopsy under fluoroscopic control or by surgical biopsy. X-ray therapy caused the tumor to become smaller, but it did not disappear. Therefore the remainder of the tumor was removed surgically. In the eighteen months since operation no recurrence has been noted.

REFERENCES

ARONS, I. Mediastinal tumors and malignant lymphoma. *Radiology,* 26: 605–621, 1936.
PFAHLER, G. E. Personal communication.
PFAHLER, G. E. Roentgen diagnosis of mediastinal tumors and their differentiation. *Am. J. Roentgenol.,* 31: 458 (April) 1934.
MULLER, G. P. Tumors of the mediastinum caused by Hodgkin's disease. *Ann. Surg.,* 105: 177 (Feb.) 1937.

GAS GANGRENE OF THE ABDOMINAL WALL FOLLOWING RUPTURED GANGRENOUS APPENDICITIS

PHILLIP E. SMITH, M.D., F.A.C.S.   AND   JOSEPH G. ZIMRING, M.D.

Attending Surgeon, Bushwick Hospital   House Surgeon, Bushwick Hospital

BROOKLYN, NEW YORK

GAS gangrene of the abdominal wall following appendicitis is a rare condition and very serious in nature. The acute, fulminating toxic type of gangrene, if not recognized early, rapidly proves fatal.

Almost all of these infections following abdominal operations are the result of the presence of the organism in the pathologic process or in the intestinal tract. The most common organism found is the Bacillus welchii.

Brown and Murphy,[1] in reporting a case of progressive postoperative gangrene of the abdominal wall following an appendectomy, state that Lynn of Baltimore has seen or collected from literature up to November, 1931 only fourteen such cases.

Gamble[2] reports eight cases of gas gangrene of the abdominal wall in 429 cases of appendicitis in which drainage was used. There were five deaths.

Shipley[3] reported a case of progressive ulceration of the abdominal wall which evidently followed drainage of a walled-off appendiceal abscess. The patient recovered after treatment with cautery and grafts.

Shearer[4] emphasizes the fact that gas gangrene of the abdominal wall responds to wide incision and drainage in enough cases to warrant the radical procedure, provided, of course, the patient's general condition will permit the treatment. His case followed gangrenous appendicitis; crepitus was felt forty hours postoperatively and a smear and blood culture showed pure cultures of Bacillus welchii. The patient died forty-four hours after operation.

Howe[5] reported a case following an operation for chronic appendicitis. The treatment consisted of wide opening of the abdominal wound, insertion of a drain to the peritoneum and administration of antitoxin. The Welch bacillus was not isolated. The patient received 180,000 units of gas gangrene antitoxin and recovered.

CASE REPORT

This case represents the sole instance of gas gangrene of the abdominal wall among 691 cases of acute and gangrenous appendicitis at the Bushwick Hospital (1934–1938).

The patient, a white male, aged 32, was admitted January 13, 1938 with complaints of severe generalized abdominal pain, nausea and vomiting. These had been present for three days, during which time the patient had taken neither solid nor liquid food. The pain had begun in the region of the epigastrium and had then passed to the lower right quadrant. Each attack of pain was accompanied by a feeling of warmth.

Physical examination showed that the patient was dehydrated, with dry, coated tongue. The abdomen was board-like, showed direct and rebound tenderness and there was a positive Blaisdell sign. No masses were felt. Acute ruptured appendicitis with peritonitis was diagnosed.

The white blood count was 29,950, with 90 per cent polymorphonuclear cells. The urine was positive for acetone.

After the administration of 1,000 c.c. of 5 per cent glucose in saline intravenously, the abdomen was opened. An acute gangrenous ruptured appendix with local peritonitis was found. The omentum was plastered around the appendix and both were lying over the urinary bladder. Three inches of the tip of the omentum were gangrenous. The peritoneum was thickened and indurated. No free fluid was present in the abdominal cavity. The appendix was removed with the actual cautery and the gangrenous tip of the omentum was removed by ligation and excision. The abdominal wound was closed in layers without drainage. Another

intravenous injection of 2,000 c.c. of 5 per cent glucose was given.

For two days postoperatively the patient's condition was fair and seemed to improve. His temperature dropped from 104 degrees to 101.6. Dehydration was still present and he appeared definitely toxic. Intravenous glucose was continued. On the third day, the patient vomited dark, greenish fluid and complained of abdominal pain. Twenty-four hours later two tension sutures and two skin clips were removed from the wound and a foul-smelling, dark fluid escaped from its lower edge. The skin was dark and crepitus could be felt on both sides of the wound. The temperature was 104.4°F., the pulse 140 and the respirations 30 per minute.

Aerobic and anaerobic cultures were taken from the wound and showed Cl. welchii. Five thousand units of gas bacillus and tetanus antitoxin were given intravenously immediately, followed by 23,700 units of polyvalent gas gangrene antitoxin. A Wangensteen drip apparatus was used because the patient continued to vomit.

On the following day, the fifth after operation, the patient was delirious and his condition definitely worse. The wound still discharged foul-smelling fluid and crepitation was advancing to the chest and groin. An additional 23,700 units of polyvalent gas gangrene antitoxin were given intravenously. However, the temperature remained high, the pulse rate went to 150 and then to 160, respirations advanced to 44 per minute, and the patient's condition was critical. The dose of polyvalent gas gangrene antitoxin was repeated in the afternoon, but shortly after midnight he died.

No post-mortem examination could be done. However, an x-ray report, made on the fourth day after operation, showed gas infection of the abdominal wall, obstruction in the upper ileum and peritoneal effusion. The pathologic report covering the specimens removed at operation showed an acute, gangrenous, ruptured appendix, an omentum dark red in color and covered with a plastic grayish exudate.

CONCLUSIONS

1. The acute fulminating toxic type of gas gangrene of the abdominal wall following appendicitis, as illustrated by this case, is a rare and rapidly fatal condition.

2. Polyvalent gas gangrene antitoxin (71,100 units) and 5,000 units of gas bacillus and tetanus antitoxin were of no avail.

3. Multiple wide incisions over the areas of crepitation, as advocated by Shearer,[4] were not made.

REFERENCES

1. BROWN and MURPHY. Progressive postoperative gangrene of the abdominal wall following appendectomy. *J. A. M. A.*, 101: 1876–1878 (Dec. 9) 1933.
2. GAMBLE, H. A. Emphysematous gangrene of the abdominal wall. *Am. J. Surg.*, 28: 389–406 (May) 1935.
3. SHIPLEY, A. M. Progressive ulceration of the abdominal wall. *Ann. Surg.*, 87: 245–249 (Feb.) 1928.
4. SHEARER. Gas gangrene following appendicitis. *Ann. Surg.*, 90: 1114–1117 (Dec.) 1929.
5. HOWE. Report of a case of gas bacillus infection occurring in wound following operation for chronic appendicitis. *New England J. Med.*, 215: 871 (Nov. 5) 1936.

A PNEUMATIC DYNAMOMETER

EDWIN O. GECKELER, M.D., F.A.C.S.

Lecturer in Surgery, Hahnemann Medical College and Hospital

PHILADELPHIA, PENNSYLVANIA

INJURIES to the upper extremity are often followed by weakness and disability of the hand. In determining the individual's ability to work it is necessary to evaluate the strength of the hand, as the grasping power is of chief importance in the function of the entire limb.

In order to measure this disability accurately, the writer has devised a pneumatic form of dynamometer which has been used with much satisfaction. This instrument combines an air gauge and blood pressure bulb connected with rubber tubing. Its soft rubber bulb is easily squeezed even in tender or partially stiff hands, and there is a sufficient range in the calibrations to register from a feeble to the strongest grasp. The gauge is calibrated both in pounds and in percentages.

For examinations the escape valve is tightened, thus holding the indicator at the maximum figure. Such an apparatus is also helpful for daily exercises, as the injured person can note his progress and thus is encouraged during recovery. For exercising, the valve is kept open, so that the needle oscillates.

The satisfactory results obtained with this dynamometer in the examination of industrial cases suggests its use also in neurology and athletics.

FIG. I.
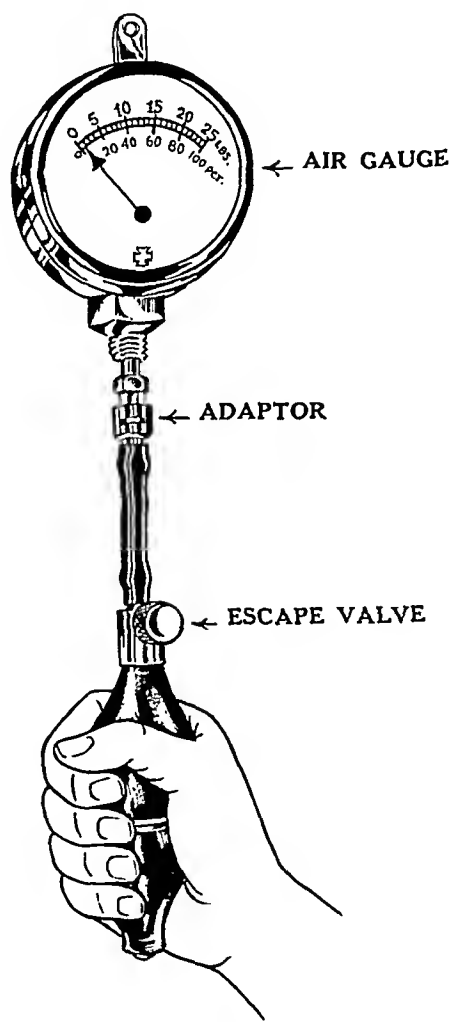
A NEW TYPE OF BLOOD TRANSFUSION APPARATUS*

SAMUEL PENNELL, M.D.

BROOKLYN, NEW YORK

THE instrument[1] herein described operates on a new principle and is so simple and reliable that with its use the direct transfusion of whole blood becomes a trouble-free procedure.

The transfused blood passes from the donor's vein to the recipient's vein through a short piece of rubber tubing and never comes in contact with any part of the instrument.

The instrument is constructed entirely of metal, has no breakable parts and fulfills all the factors of safety required of a transfusion apparatus.

Principle. A piece of rubber tubing (C) lies in a channel on top of a large worm (D), as shown in Figure 1. The tubing is held against this worm by means of a bar (B) so that it is pinched at the points where it comes in contact with the worm (A). As the worm rotates its edges slide along the rubber tubing. This creates suction at one end of the tubing and pressure at the other. Continued rotation of the worm causes a continuous stream of blood to flow through the tubing.

The amount of blood delivered depends on the inside diameter of the tubing. The tubing has therefore been calibrated and a counter on the instrument records the amount of blood delivered in CC.

The instrument is small, compact and of all metal construction. (Fig. 2.) It may be sterilized by boiling. It has a heavily weighted base (O), and will stand upright wherever it is placed. The base is attached to the instrument proper by means of a bayonet fitting so that with one twist, the base can be removed and the instrument held in the hand if so desired by grasping the upright (L).

A handle (M) is provided by means of which the worm (D) is rotated. *The handle can be turned only one way. This allows the blood to flow in one direction only and there can be no back-flow.*

A counter (G) which records the amount of blood in CC is mounted so that it can easily be read.

At the top of the instrument is a flat metal cover (B) held in place by two spring clips (F). Removal of the cover reveals the channel in which the tubing lies when the instrument is in use. Two pieces of glass tubing, a straight piece (J) and a T-tube (H) serve as observation windows.

For Transfusion. A few simple steps make the instrument ready for a transfusion. The cover is removed, 20 c.c. of glycerin is poured into the chamber around the worm, the tubing is placed in the channel and the cover is replaced.

Rotation of the handle while the donor's side of the tubing is immersed in saline will fill the tubing with saline. The transfusion may then be given.

Although the instrument is boilable, only the tubing need be sterilized unless the transfusion is to be given in a completely sterile set-up. Since the blood passes only through the tubing, never coming in contact with the instrument, a previously sterilized tubing, if always on hand, makes the instrument immediately available in an emergency.

Transfusion Statistics. During its development the instrument has been used in over 300 transfusions at the Kings County Hospital and in about 175 at the Brooklyn Jewish Hospital. All were given with uniform ease. There has been no clotting even though as much as 750 c.c. of blood was given on several occasions.

---

[1] The instrument is manufactured by Fred Haslam & Co., Inc., Brooklyn, New York.

In the 175 transfusions given at the Brooklyn Jewish Hospital, only two mild post-transfusion chills and one mild urticarial reaction were reported.

blood through the apparatus fifteen times, but no red cell destruction was observed.

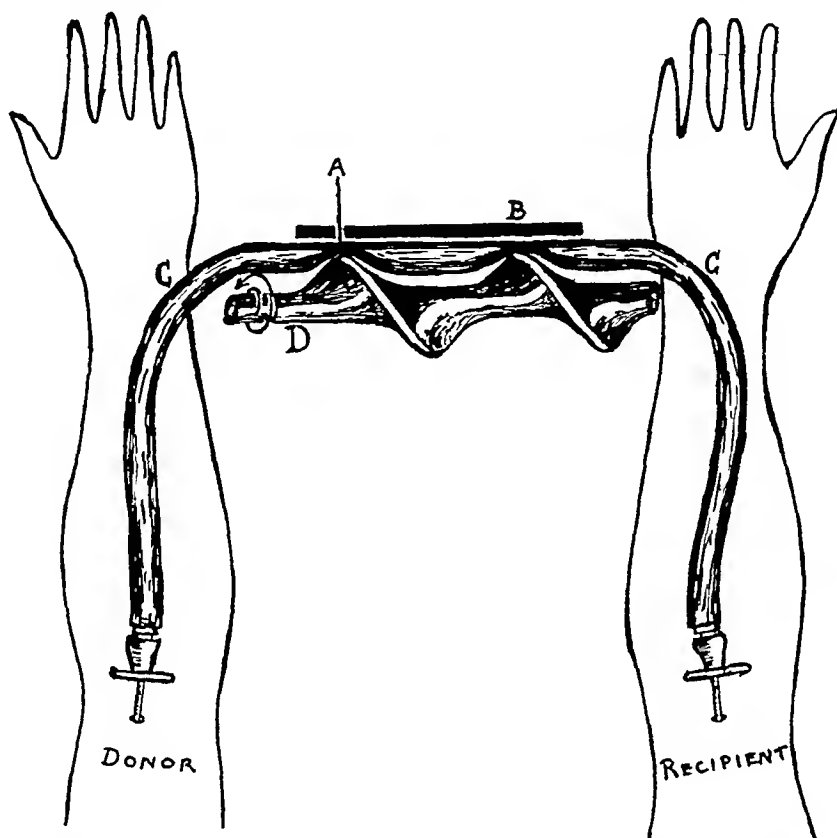*Other Uses.* The instrument, although primarily a transfuson apparatus, lends it-

FIG. 1. A, point at which worm pinches tubing. B, cover, C, rubber tubing. D, worm. Schematic drawing illustrating principle upon which instrument operates.
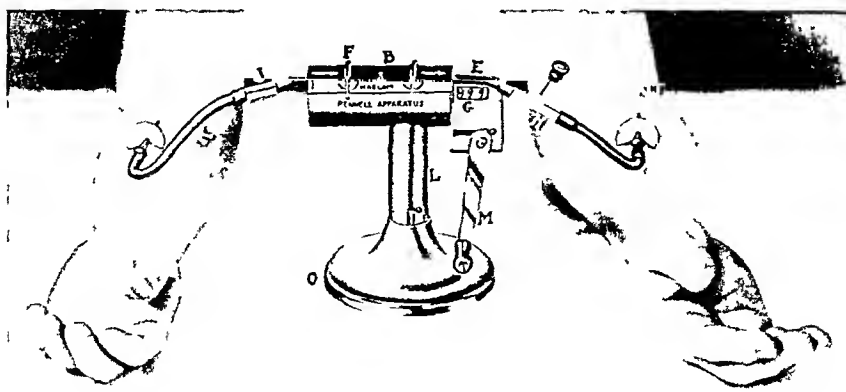

FIG. 2. B, cover. E, middle section rubber tubing (same as C in Figure 1). F, clips. G, counter. H, glass T-tube. J, glass connecting tube. K, tubing from donor. L, upright stand. M, handle. N, tubing to recipient. O, removable weighted base.

Dr. J. R. O'Sullivan of Kearny, New Jersey ran a quantity of citrated human

self to the following other medical uses and in each instance simplifies the procedure:

1. *Phlebotomy*. The instrument can be taken to the bedside and the usual messiness of phlebotomy eliminated. It also provides a simple means of collecting the blood in a sterile manner by inserting the delivery end of the tubing into a sterile flask containing citrate. The amount withdrawn is recorded by the counter.

2. *Aspiration*. By connecting the suction side of the tubing to the aspirating needle, continuous suction can be applied to chest or abdomen. The amount of fluid that can be aspirated is limited only by the desire of the operator or content of the cavity.

3. *Pneumothorax*. The instrument pumps air as well as blood. By attaching the pneumothorax needle to the delivery side of the tubing, and the upright limb of the glass T-tube (*H*) to the water manometer, one has an efficient pneumothorax apparatus. The necessity for water bottles is eliminated. The air may be pumped in as slowly as desired under manometric control and the amount introduced is recorded by the counter.

SUMMARY

A blood transfusion apparatus is described which operates on a new principle. It has no syringes or valves. It has the advantages of being very simple in operation, reliable, unbreakable and extremely efficient in the handling of whole blood. It is versatile and lends itself to other uses besides transfusion such as phlebotomy, aspiration and pneumothorax.

Formation of Pelvic Viscera and Their Outlets on the Surface

BY

EDWARD LEVY, M.D.

Assistant Attending Surgeon, Polyclinic Medical School and Hospital;
Associate Visiting Proctologist, Cancer Institute

FORMATION OF PELVIC VISCERA AND THEIR OUTLETS ON THE SURFACE*

EDWARD LEVY, M.D.

Assistant Attending Surgeon, Polyclinic Medical School and Hospital; Associate Visiting Proctologist, Cancer Institute

NEW YORK CITY

PART I

MATURATION—FERTILIZATION—ZYGOTE—EMBRYO TO FORMATION OF INTERNAL CLOACA

THE anatomic history of an individual vertebrate begins with the life history of its parental germ cells. These cells are early laid aside as gonocytes in a small strip of mesothelium on the ventromedial border of the urogenital folds. Here the gonocytes proliferate extensively and become lodged in their differentiated sex glands as the female sex cells or oögonia of the ovaries; the male sex cells or spermatogonia of the testes. The gonocytes during their active stage of multiplication resemble the body cells—soma cells—in their structural appearance and in their mitotic process of equational chromosome division in which the number of chromosomes remains constant for each cell. The primordial sex cells eventually cease multiplying and enter upon a stage of growth in preparation for two specialized and final divisions. The descendants of the gonocytes that have attained the period of growth—primary oöcytes in the ovaries, and primary spermatocytes in the testes—realign their paired chromosomes, one derived from each parent, in single segments whose number is half that of the chromosome number.

The primary spermatocyte divides into two equal and smaller secondary spermatocytes, each of which acquires one of the paired chromosomes forming each segment. By this reductional division— miosis—one half of the chromosomes has been eliminated from each of the two secondary spermatocytes, and the sex chromosome resides in but one of the two secondary spermatocytes. The two

First Cleavage

Fertized Ovum

Falloppian Tube

Many cell stage

Morula stage

Uterine Cavity

Beginning of implantation of Ovum

A.

B.

C.

Implantation of ovum.

Sealing of point of entry.

D.

E.

Formation of Trophoblast.
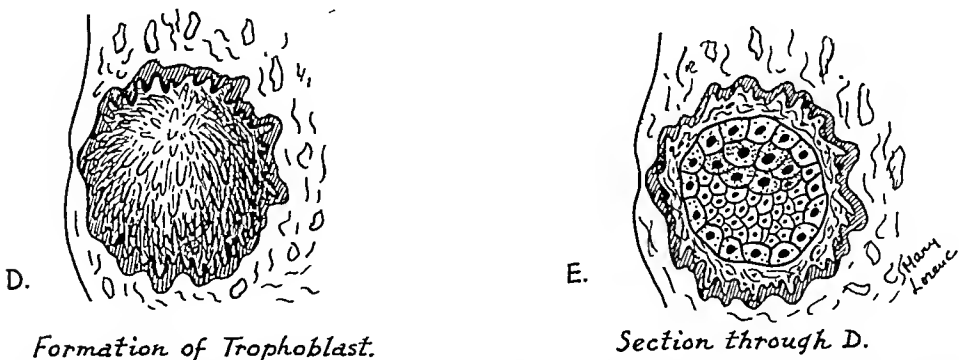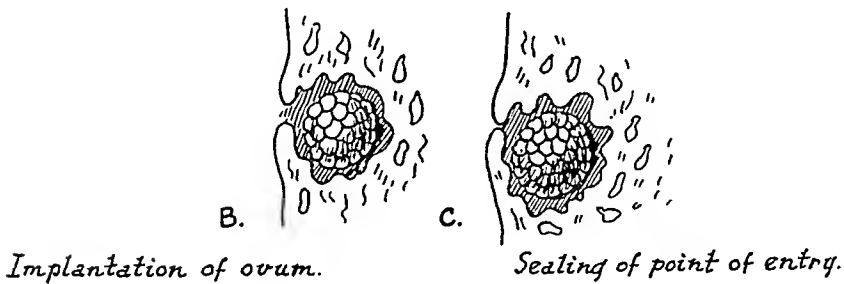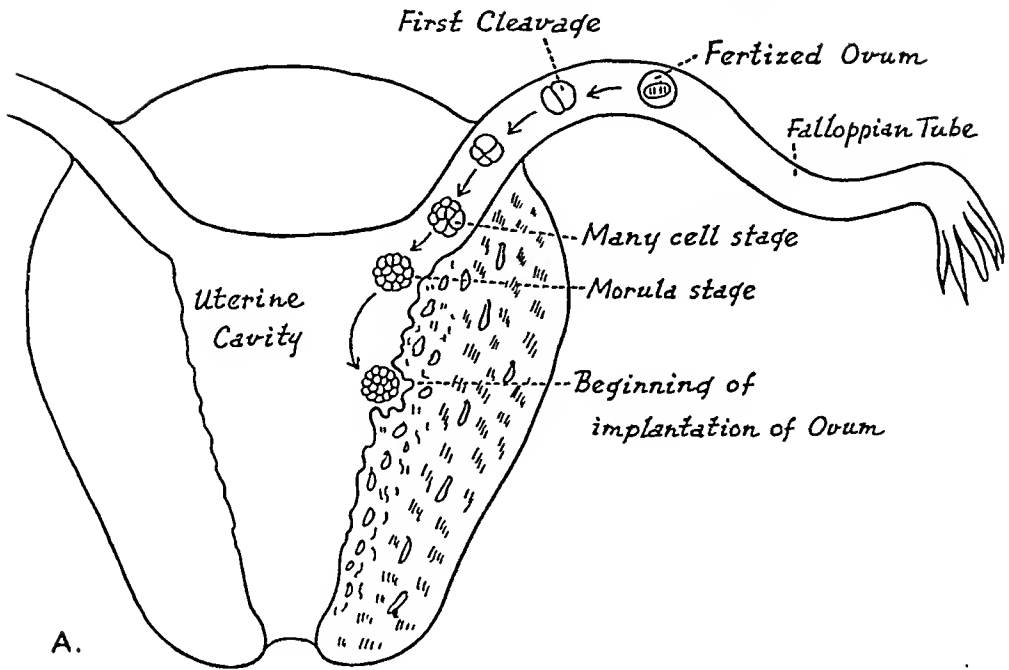
Section through D.

PLATE I. A, uterus and fallopian tube with fertilized ovum, showing cleavage and beginning implantation. B, implantation. C, sealing of point of entry of ovum. D, formation of trophoblast on wall of zygote. E, section through D to show inner cell mass.

secondary spermatocytes undergo the final second division by equational splitting of their chromosomes and yield four equal smaller spermatids of which two possess the sex chromosome and each contains half the number of chromosomes fixed for the species. The spermatids are each transformed into a spermatozoon by a complex process which provides the matured male sex cell—male gamete—with a locomotor apparatus. The spermatozoon has the appearance of a lance 0.05 mm. long.

The primary oöcyte divides into two markedly unequal cells by the same reduction process that took place in the primary spermatocytes. The very small cell—first polar body—contains half the chromosomes and little cytoplasm; the large cell—secondary oöcyte—contains half the chromosomes and almost all of the parent cytoplasm. Each of the two cells undergoes the final second division by mitosis and four cells are formed. The first polar body divides into two cells; the secondary oöcyte divides into a small cell—the second polar body—and a large cell—female gamete—the matured ovum. Each of the four cells contains one half the number of chromosomes fixed for the species. Of these four cells only the matured ovum, after fertilization, is capable of developing a normal individual. Fertilization of the polar bodies may give rise to monster formations. The female gamete is a spheroid body whose greatest diameter is 0.25 mm.

The ovarian follicle containing a secondary oöcyte, distends, and upon rupturing liberates its oöcyte which is then guided by the ciliary current of the oviduct down its lumen. In the outer portion of the tube, a spermatozoon pierces the outer layer of the secondary oöcyte and enters its cytoplasm. The secondary oöcyte gives off its second polar body and becomes the mature ovum whose nuclear material forms the female pronucleus. The head of the spermatozoon, practically all nuclear material, is transformed into the male pronucleus which is accompanied by the two centrosomes that reside in the middle piece, and the flagellum is absorbed. The male pronucleus is smaller than the female pronucleus. Each pronucleus contains one half the number of chromosomes typical for the species.

The male and female pronuclei fuse and form a single nucleus—segmentation nucleus—and fertilization is completed. The cell resulting from the union of the male and female gametes is the fertilized ovum—zygote—to which the full number of chromosomes fixed for the species has been restored. The zygote has the capacity of developing into a new individual of the species, whose sex is determined by the chromosome constitution of its nucleus. Through
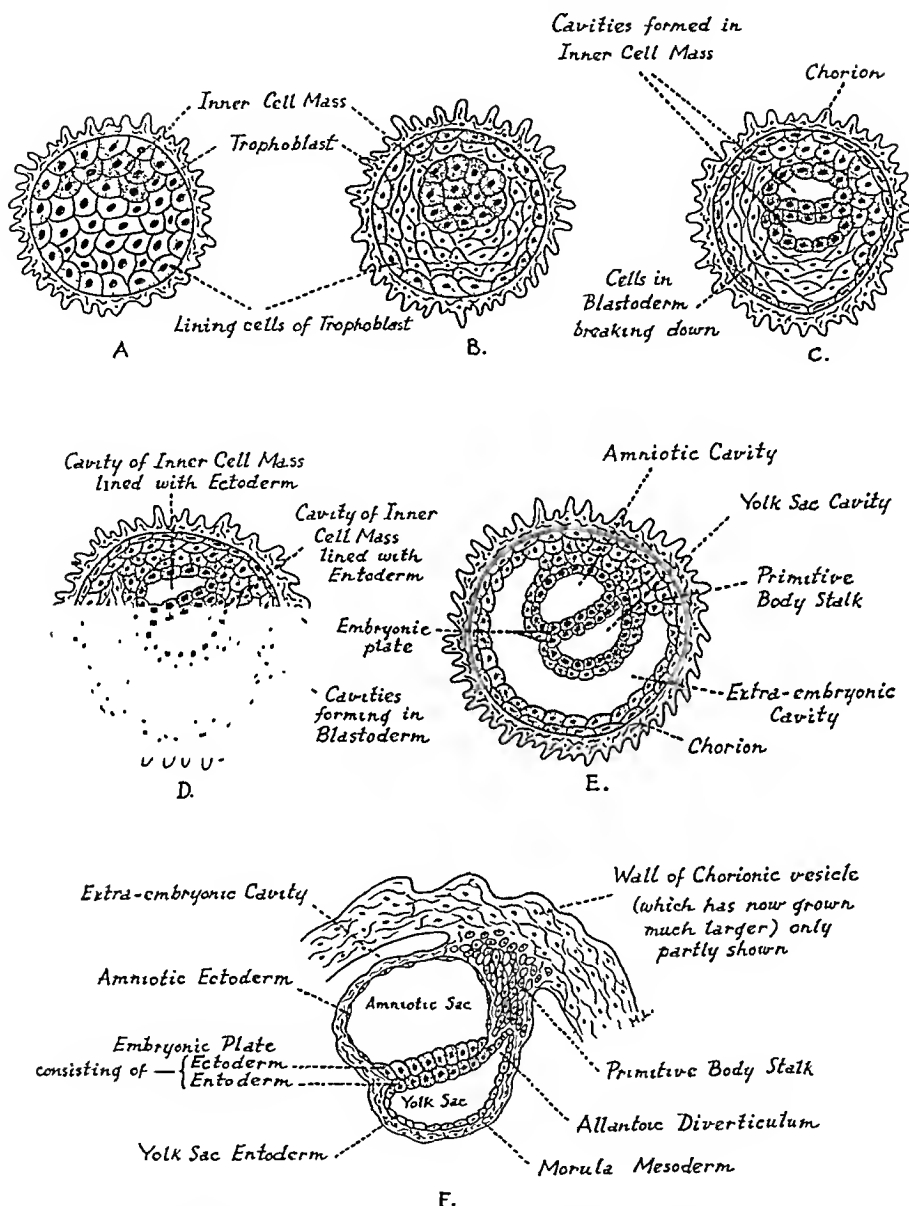
PLATE II. A, same as E in Plate I. Zygote, showing inner cell mass and surrounding tissue. B, zygote lining cells of trophoblast. Inner cell mass separated from wall of blastoderm. C, cavities formed in inner cell mass, surrounding cells in blastoderm breaking down. D, cavities forming in morula mesoderm of blastoderm. E, formation of primitive body stalk. Amniotic and yolk sacs, and completed extra-embryonic cavity. F, identification of primitive structures; the embryonic plate with its cells differentiated from cells lining the amniotic and yolk sac cavities.

the genes of the chromosomes it is endowed with all the potentialities of its parents.

The zygote immediately enters upon the period of segmentation. By a series of consecutive mitotic divisions it develops into a large number of cells which are grouped together in the form of a solid spheroidal mass, the morula. Early in the segmentation process the cell cleavages appear equal in size, and later the divisions result in the formation of cells of different sizes and apparently different functions. A few cells retain all the potentialities derived from their parents and develop into the parental or primordial germ cells. To the remaining cells only parts of the chromosome inheritances are transferred and they develop into the soma cells whose descendants are the tissue cells of the body.

The zygote, scarcely changed in size during the stage of segmentation, easily reaches the cavity of the uterus, where a special cellular formation of the mucous membrane—decidual reaction—has taken place. The outer layer of the cell mass of the morula develops into a syncytial covering layer—trophoblast—which exerts a digestive action on the decidua. The uterine epithelium in contact with the zygote is destroyed and the zygote sinks into the uterine mucosa where it becomes enclosed by the mucous membrane growing over it.

Until implantation takes place, the zygote is a free organism in the uterine cavity dependent upon its own small reserves for nourishment. Consequently during this period it hardly grows. Upon implantation, the zygote becomes a parasite upon the maternal organism from which its nourishment is derived throughout the remainder of the intra-uterine period. With the establishment of the parasitic relationship the ovum enters upon a period of extremely rapid growth.

The morula cells of the zygote arrange themselves in a cluster—inner cell mass—suspended from the outer enveloping layer of trophoderm in a bed of slightly differentiated cells—morula mesoderm—developed from both inner cell mass and trophoderm. The morula mesoderm grows completely around the inner cell mass and separates the cell mass from the trophoderm. The detached inner cell mass lies in the bed of morula mesoderm.

Two clefts appear in the detached cluster of cells and expand into the two closely apposed vesicles of the future embryo, the external or amniotic vesicle and the internal or yolk vesicle. In the morula mesoderm, spaces appear and on becoming confluent form a single large cavity—extra-embryonic acvity of the embryo.

The bivesicular structure ensheathed in morula mesoderm lies in a bulge of the wall of the extra-embryonic cavity.

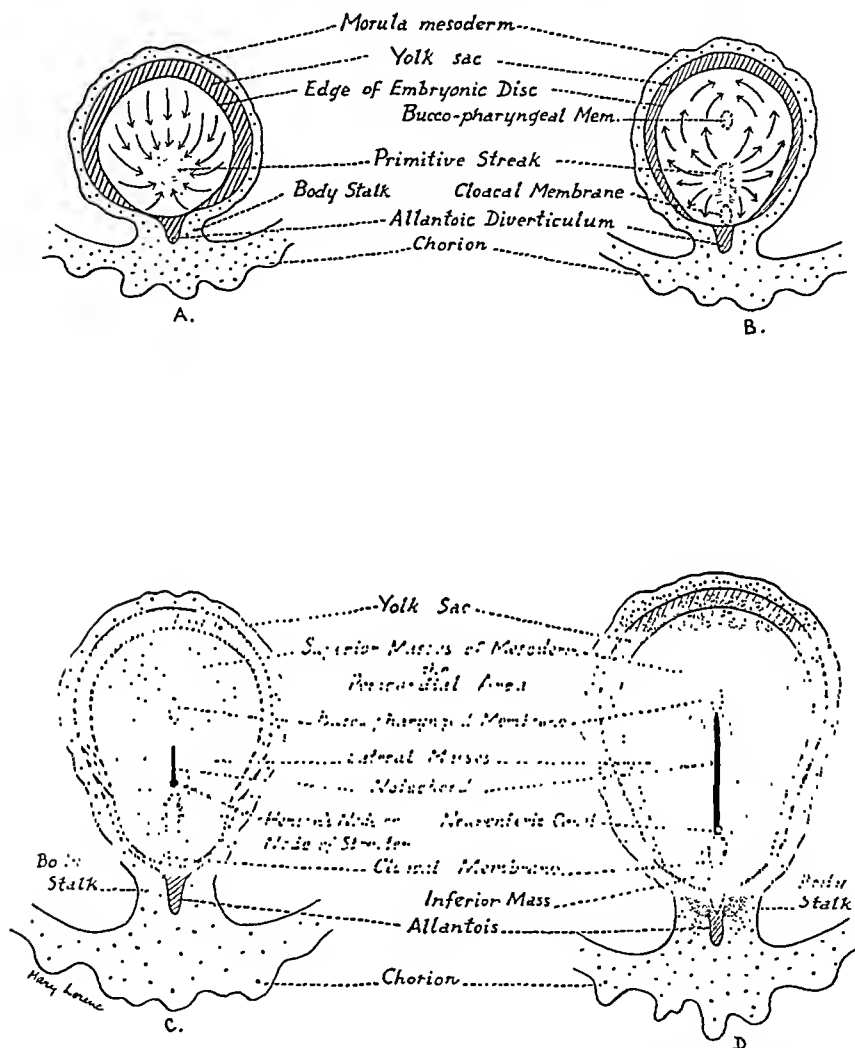The zygote has been transformed from a solid spheroidal struc-

PLATE III. Top view of embryo. A, formation of primitive streak. Direction of wave of activity. B, direction of wave of proliferation of mesoderm from primitive streak. C, mesodermic wings extending through plate, beginning of notochord. D, mesodermic wings complete, notochord complete. Neurenteric canal.

ture into a large vesicle containing two smaller ones in its medial wall. Each of the two small vesicles is lined on its internal surface by a continuous layer of cells. The linings of the two vesicles are applied to each other in the small area where the vesicles are apposed, and form the bilaminar disc, shield, or plate of the embryonal

area.* The external or amniotic vesicle is lined with ectoderm and its floor is the dorsal lamella of the embryonal disc. The internal or yolk vesicle is lined with entoderm and its roof is the ventral lamella of the embryonal disc. The ventral lamella of entoderm continues as the lining of the primitive alimentary tract and its appendages. The dorsal lamella of ectoderm provides the material for the development of the remainder of the embryo.

The bivesicular structure lies in a sheath of morula mesoderm— the primitive body stalk—that extends from the inner wall of the zygote. Orientation of the embryonal disc is determined by the portion that is attached to the body stalk which is the caudal, posterior, or lower region; the portion nearest the extra-embryonic cavity is the cephalic, anterior, or upper portion. The borders between the anterior and the posterior margin are the lateral margins.

The growing disc of the embryonic area becomes pear-shaped with the appearance of a linear groove—primitive groove—with low marginal ridges in the posterior fourth of the external surface. The area marked off by the primitive groove and its marginal ridges constitutes the primitive streak and is the zone of intensely active division of the cells of the ectoderm from which the embryonic mesoderm is formed by proliferation and migration. The embryonic mesoderm, the ectoderm and the entoderm comprise the three germinal layers of the embryo. Posterior to the primitive streak and further caudad the plate including its junction with the body stalk is composed of ectoderm closely applied to entoderm to form the cloacal membrane.

The cloacal membrane is distinct from the primitive streak and extends for a short distance on the body stalk. The growing sacks of the vesicles by their internally advancing portions stretch the intervening wall of morula mesoderm into a thin sheath lining the extra-embryonic cavity; the externally advancing portion of the amniotic sac reduces the broad base of the primitive body stalk to the narrower body stalk. The external portion of the growing yolk sac invades the body stalk by a slender diverticulum—allantois whose wall is partly bound by the cloacal membrane on the body stalk.

---

* The embryonal area of the zygote will form all of the future individual. The remainder of the zygote enters into the formation of the membranes and appendages which provide protection, nourishment, and support during intra-uterine life, and are cast off when the umbilical cord is severed at birth.
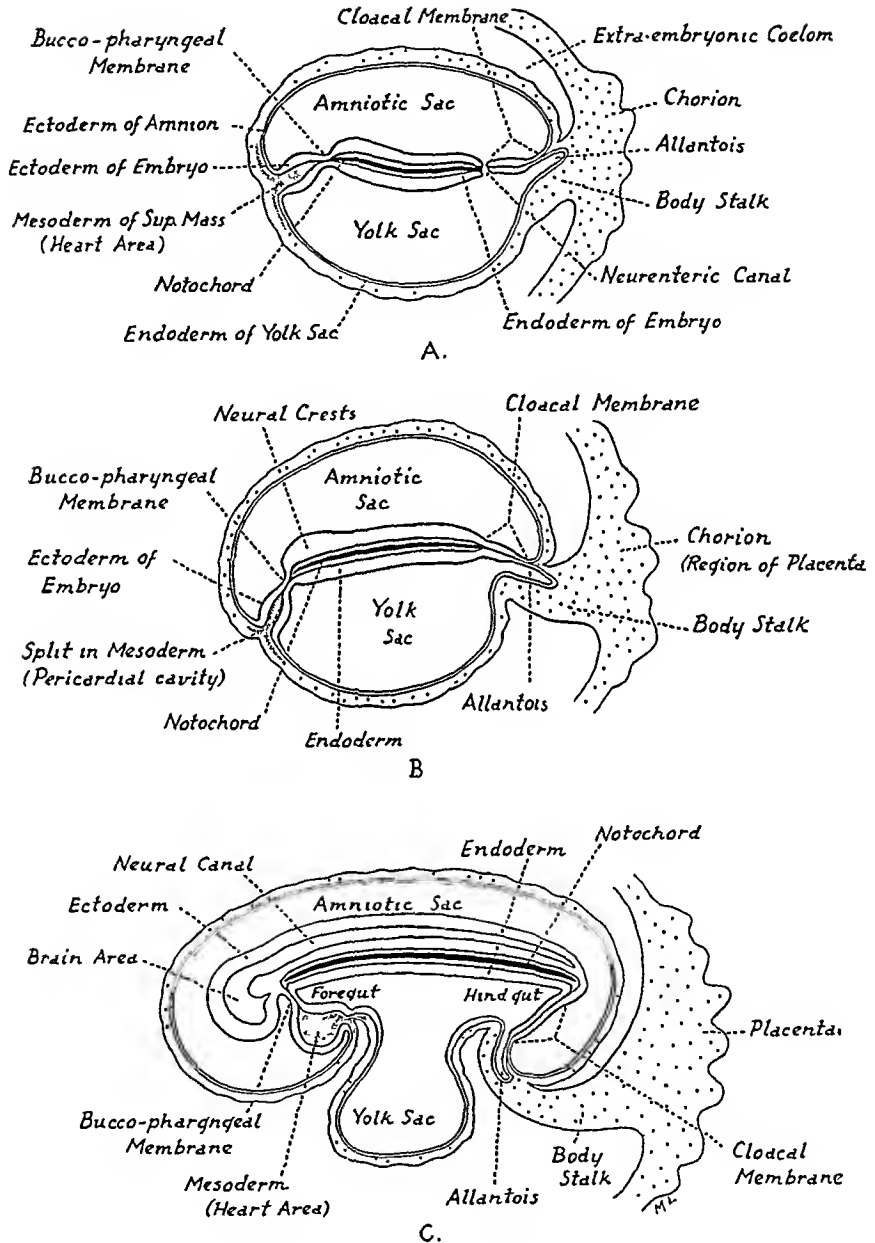
Cloacal Membrane

Bucco-pharyngeal Membrane

Extra-embryonic Coelom

Amniotic Sac

Chorion

Ectoderm of Amnion

Allantois

Ectoderm of Embryo

Body Stalk

Mesoderm of Sup. Mass (Heart Area)

Yolk Sac

Notochord

Neurenteric Canal

Endoderm of Yolk Sac

Endoderm of Embryo

A.

Neural Crests

Cloacal Membrane

Bucco-pharyngeal Membrane

Amniotic Sac

Ectoderm of Embryo

Chorion (Region of Placenta

Split in Mesoderm (Pericardial cavity)

Yolk Sac

Body Stalk

Notochord

Allantois

Endoderm

B

Neural Canal

Endoderm

Notochord

Ectoderm

Amniotic Sac

Brain Area

Foregut

Hindgut

Placenta

Bucco-pharyngeal Membrane

Yolk Sac

Body Stalk

Cloacal Membrane

Mesoderm (Heart Area)

Allantois

C.

PLATE IV. Median sagittal sections. A, embryonic plate (with neurenteric canal open) being bent gently beyond ends of notochord by pressure of amniotic fluid. B, plate bent more, neurenteric canal closed. C, plate bent still more, forming head and tail folds for foregut and hindgut, drawing in of yolk sac. Extension of cloacal membrane on stalk.

The mesodermal cells from the lateral margins of the primitive streak where their proliferation is most intense, stream into the plate between the ectoderm and entoderm and condense into paired sheets. The lateral sheets grow into the plate in a paired wing-shaped formation and extend through the margin of the plate to fuse by their peripheral borders with the morula mesoderm that invests the bivesicular structure. The anterior crescents of the embryonal mesodermal formations meet cephalad to the bucco-pharyngeal membrane and fuse in a wide band to form the proto-cardiac area. The posterior crescents extend downward along each side of the cloacal membrane to the body stalk where they fuse with the morula mesoderm of the stalk. With the development of the embryonal mesoderm, the zygote becomes the embryo.

In a circumscribed area adjoining the cephalic end of the primitive streak—node of Streeter or Henson's knot—the proliferated cells from the ectoderm fuse with the underlying entodermal cells and enter upon an active state of multiplication. The cells form a column that penetrates the plate between the ectoderm and ento-derm and fuses with the latter. The column occupies the space between the unfused crescent of the mesoderm formation up to the buccopharyngeal membrane. The mesodermal cells of the paired sheets are closely applied and distinct from this axial formation—notochordal process or head process. The developing notochordal process fuses with the underlying entodermal cells and draws away from the overlying ectoderm which sinks down and forms a groove—neural groove. The bulky mesoderm at each side of the notochord raises the margins of the groove in the overlying ectoderm which form the neural ridges or folds. The growth of the neural structures coincides with the growth of the notochord. The neural folds close the neural groove anteriorly by joining in a low terminal ridge which separates the groove from the buccopharyngeal area. Posteriorly the elevated folds diverge and extend into the prominent masses at each side of the primitive streak and embrace in the angle of their diverging margins the anterior portion of the streak and the noto-chordal node which become included in the neural floor. The noto-chordal process grows forward in a rod of multiple layered cells which are fused with the underlying entoderm. Cephalad, the layers of the rod become gradually reduced and fade into the entoderm immediately posterior to the fused ectoderm and entoderm of the buccopharyngeal membrane. A lumen appears in the notochordal process and rapidly forms a channel—canal of the notochord—which opens posteriorly, caudal to the notochordal node on the
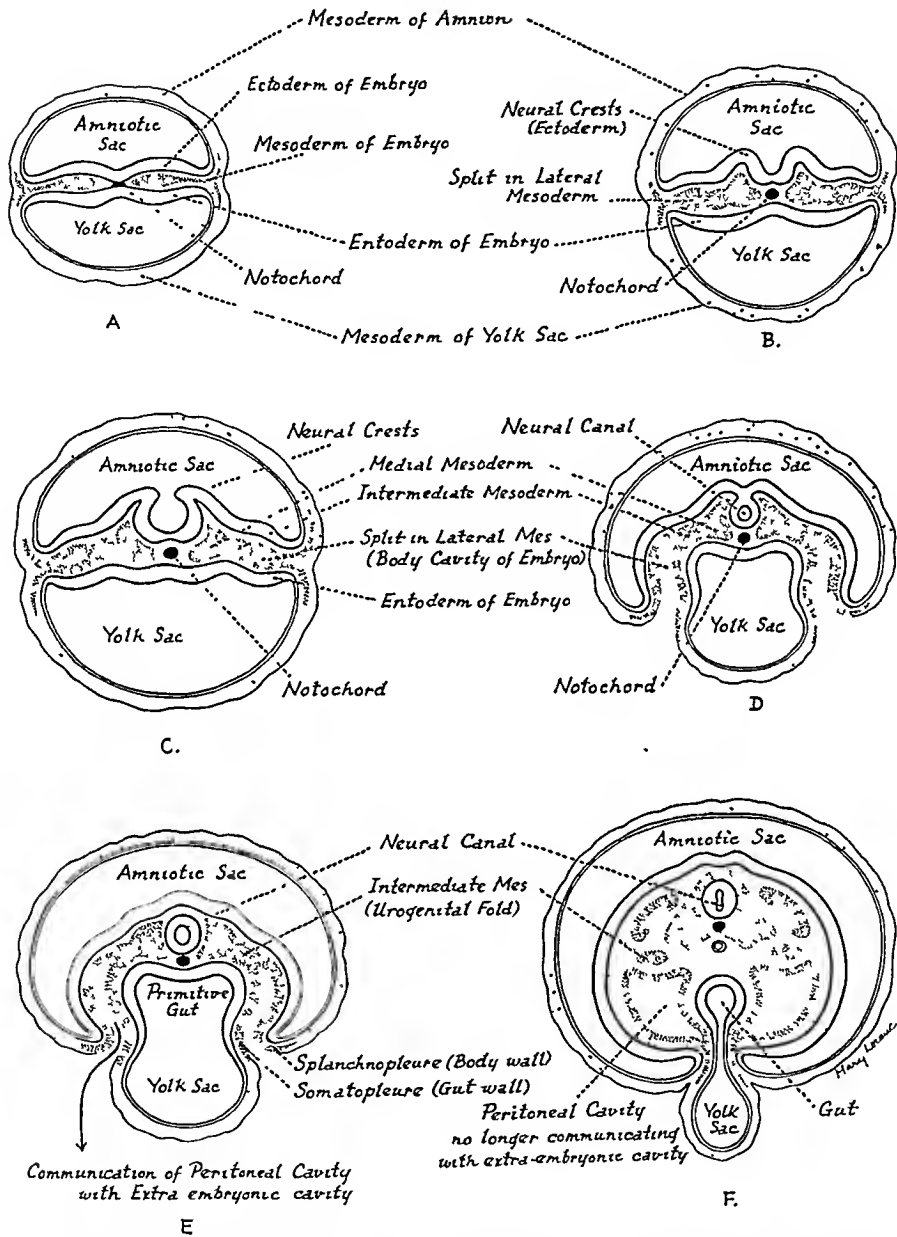
PLATE V. Transverse sections A, embryonic plate. Ectoderm, entoderm and mesoderm. B, arrangement of three parts of mesoderm; beginning of split in lateral sheets. Rising of neural crests. C, larger split in lateral mesoderm, neural crests higher. D, embryonic plate bending over yolk sac, split in lateral mesoderm complete, forming open body cavity of embryo. Neural crests closed, forming neural canal. E, plate bent still more, forming primitive gut; yolk sac enclosed in amniotic folds, identification of splanchnopleure and somatopleure; body cavity still open to extra-embryonic cavity. F, plate folded so that lateral edges meet in ventral midline, squeezing off yolk sac to narrow communicating canal. Body cavity closed off from extra-embryonic cavity.

floor of the neural groove, into the amniotic vesicle. The canal of the notochord is separated from the cavity of the yolk sac by the floor of the canal that has fused with the entodermal roof of the yolk sac. Areas of this fused canal floor and entodermal roof break down and the canal which opens into the amniotic cavity by a single external orifice in the floor of the neural groove acquires openings in the cavity of the yolk sac. The yolk sac cavity is brought into communication with the amniotic sac by the persisting portion of notochordal canal—neurenteric canal—whose external orifice is on the floor of the medullary groove in the angle of the diverging neural ridges. The notochordal remnant—chordal plate—appears as a groove—notochordal groove—on the roof of the yolk sac. The entodermal cells from each side of the groove proliferate and meet each other to reform the cylindrical tube. The notochordal canal and its extension, the neurenteric canal, are quickly obliterated by the overgrowth of neighboring cells and the notochord is converted into a solid rod of cells. The cells of the neurenteric canal might be regarded as the rudimentary tissue of the future tail of the embryo.

The primitive streak becomes effaced by the caudad growth of the notochord. The posterior termination of the notochord extends to the upper end of the cloacal membrane between the

DERIVATIVE OF THE MESODERM

Paraxial	Intermediate	Lateral Mass
Sclerotome provides hard skeletal structures of the vertebral column and its ligaments, dura mater of brain and spinal cord. Myotome forms voluntary muscle.	Upper portion is faintly marked by transverse divisions and lower portion unsegmented as nephrogenic cord. Forms urinary apparatus except urethra and most of bladder. Genital glands and their ducts.	Precipitates out mesenchyme. Lining mesothelial cells of great serous cavities; pleural, pericardial, peritoneal. Connective tissue except vertebral column and head. Mesoderm of limbs. Probably all unstriped muscle fibers of wall of alimentary canal and blood vessels. Many cells, irregular in form and possessing ameboid activity appear between the definite layers of mesoderm and adjacent ectoderm and entoderm. These are the cells of the mesenchyme and they are derived from the mesoderm and possibly the ectoderm and entoderm. These cells have a definite reaction to certain dyestuffs.

medial origins of the crescentic margins of the inferior wings of mesoderm that border the cloacal membrane.

Each sheet of mesoderm lateral to its medial border is indented by a sagittal groove which extends through its length. The medial portion of the mesoderm is divided by a series of horizontal deep grooves into successive segments or somites. Each sheet is defined into three portions; a medial or paraxial portion that lies between the groove and the notochord; an intermediate region of the sagittal groove and the lateral cell mass that extends from the groove laterally to the margin of the plate where the embryonal mesoderm becomes continuous with the morula mesoderm of the wall of the extra-embryonic cavity.

Embedded in a knob of morula mesoderm the bivesicular structure of the zygote comprising two vesicles and the oval plate of their apposed surfaces has developed into the more highly differentiated bivesicular structure of the embryo. The amniotic sac growing against the dorsum of the broad base of the primitive body stalk has narrowed the stalk which becomes elongated. The yolk sac has extended into the body stalk to form the diverticular process of the allantois.

The bilaminar oval plate of the zygote has become incompletely trilaminar with the distribution of the embryonic mesoderm that is absent in the regions of the buccopharyngeal and cloacal membranes. The oval plate has become elongated by the development of the axial structures of the solid notochord and overlying heavy neural tube in the mid-longitudinal line. The plate has the form of a violin and suggests the adult form of the trunk with the pelvis. The differentiated plate, varying in resistance with the density of the structures, opposes a varying resistance to the pressure of the accumulating amniotic fluid and to the traction of the swelling amniotic sac. The plate is most resistant in the region of the longitudinal axis occupied by the solid notochord and heavy overlying neural tube, less resistant in the transverse axis by the mesodermal masses that extend from each side of the axial structures, and least resistant at the junctions of the anterior and posterior ends of the solid notochord with the fused ectoderm and entoderm of the buccopharyngeal and cloacal membranes where there is no mesoderm to strengthen the resistance to the developing pressure and traction.

The amniotic sac is attached to the dorsum of the body stalk and to the ectodermal rim of the embryonic plate which is the floor of the amniotic cavity. The accumulating fluid of the amniotic
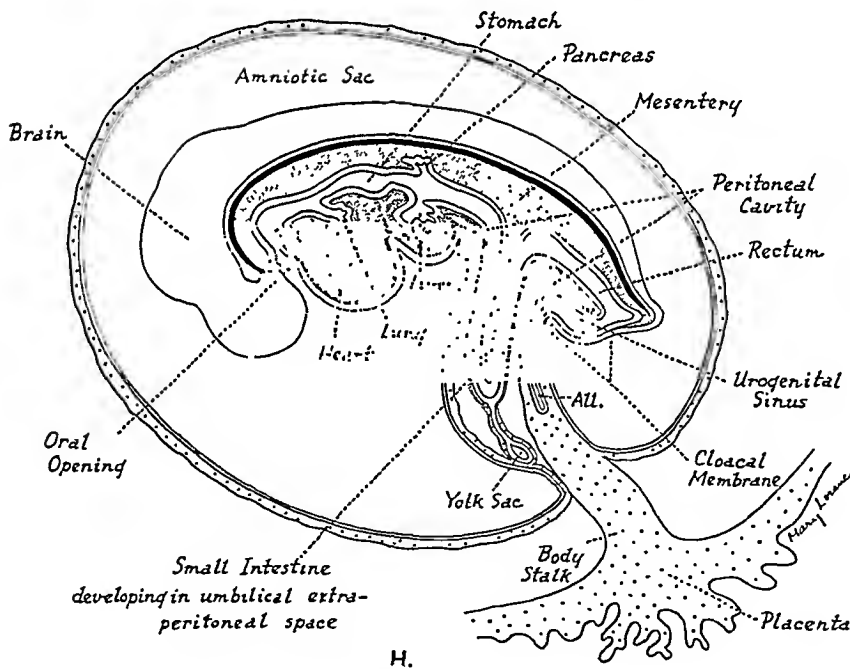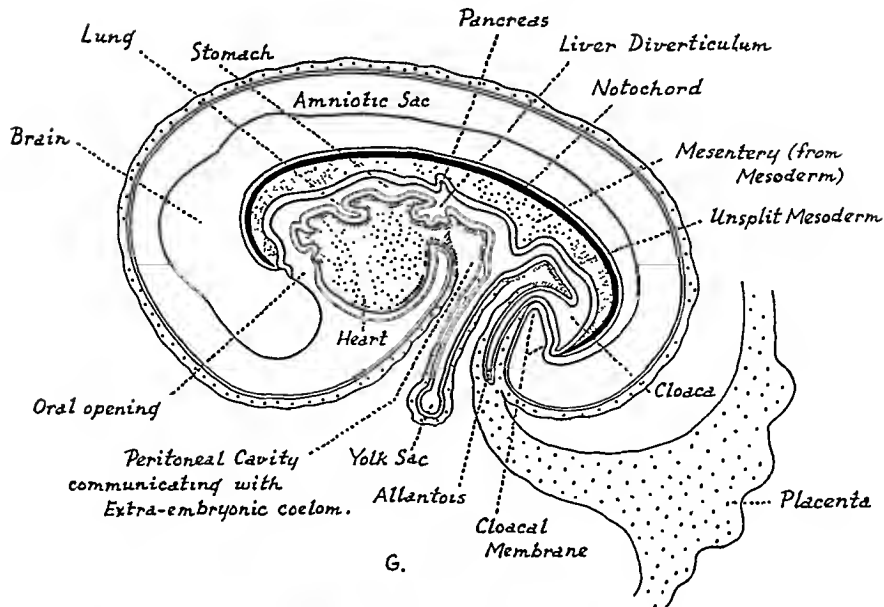
G.

H.

PLATE VI. Continuation of plate V. Median sagittal sections. G, formation of alimentary canal with its various diverticuli, formation of cloaca, lengthening body stalk—body cavity still open to extra-embryonic cavity. H, further development of alimentary canal. Small intestine forming in umbilical, extra-peritoneal space. Cloaca partly divided into urogenital cloaca and rectal cloaca.

cavity distends the rapidly growing sac which bulges beyond its attachments to the plate and stalk. The amniotic sac expands in a plane ventrad to the plate and advances medially with its attachments. The plate yields to the increasing weight of the amniotic liquid on its back and to the traction of the enveloping amniotic sac attached to its margins by folding ventrally at each end of the solid notochord to form the head and tail folds. Laterally, the pressure and traction form a gentle arching of the plate whose edges roll inward, taking with them the attached body stalk and yolk sac. The elongating stalk with its allantois is brought ventrocephalad to the posterior end of the notochord. The allantois forms an acute angle with the yolk sac and its lumen takes origin from the recess of the tail fold. The slowly growing yolk sac immediately beyond its attachment to the entodermal rim of the plate is compressed between the medially advancing folds of the amniotic sac to form the waist of the yolk sac—vitello-intestinal duct. The bending and arching of the embryo has transformed the flat plate into a cylindroid whose chamber is the primitive gut cavity.

The cavity of the medial portion of the primitive gut, the midgut, communicates widely in front with the yolk sac cavity through the large vitello-intestinal duct, cephalad through the anterior portal with the recess of the head fold—the foregut—and caudad by the posterior portal with the recess of the tail fold—primitive cloaca—which terminates ventrocephalad in the narrow channel of the allantois. The allantois, beginning as a diverticulum from the yolk sac, has changed its position to become the diverticulum of the primitive gut cavity.

The external surface of the tail fold between the caudal end of the notochord and the body stalk is occupied by the cloacal membrane which extends for a short distance on the under surface of the body stalk. The cloacal membrane, with its borders of mesoderm and covering of ectoderm, marks the external region or perineum of the future pelvic outlet. Caudal growth of the embryo beyond the cloacal membrane forms the tail.

Spaces appear in the mesoderm of the protocardiac area and within each lateral mass. The spaces become continuous. The spaces of each lateral mass become confluent and extend caudally as far as the inferior mesodermal wings of the tail fold to become the paired intra-embryonic spaces. The inferior mesodermal wings remain unsplit. Each intra-embryonic space extends peripherally through the mesodermal rim of the plate and the contiguous wall of morula
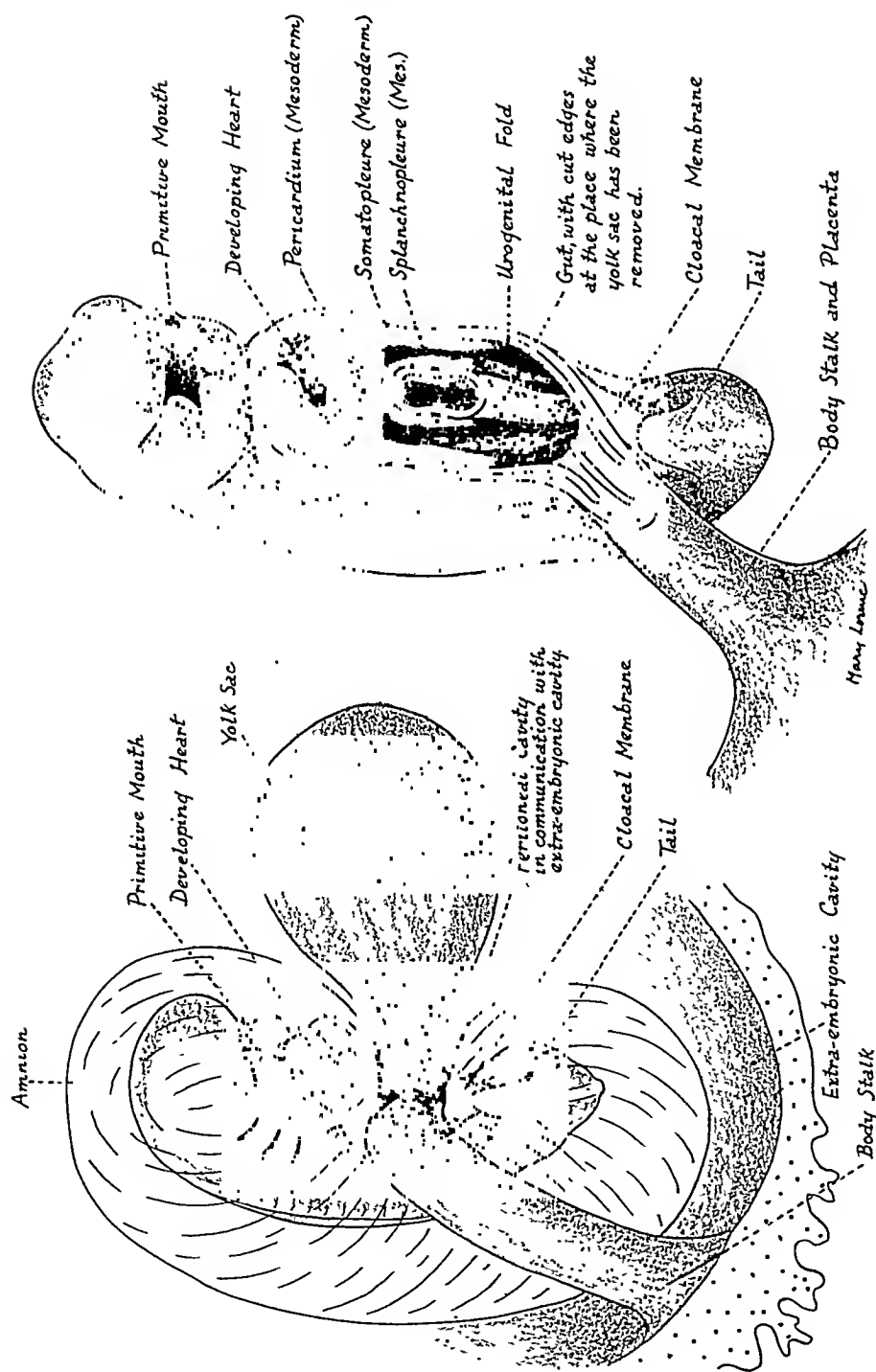
PLATE VII. External form of embryo. A, external form of embryo, enclosed in amniotic folds, body stalk and yolk sac emerging from belly of embryo. Body cavity still open to extra-embryonic cavity. B, external form, part of body wall and yolk sac cut off to show developing heart, enveloped in mesoderm, mesoderm of splanchnopleure and somatopleure, urogenital folds—peritoneal cavity.

mesoderm of the extra-embryonic cavity. The intra-embryonic space becomes continuous with the extra-embryonic space.

In the protocardiac zone the space remains confined to the mesoderm—pericardial cavity—from which paired dorsocaudal extensions—pleuroperitoneal channels—one on each side of the foregut open into the cephalic portion of each intra-embryonic space.

Each lateral mass of embryonic mesoderm is split by its cavitation process and forms two layers of mesoderm partially enclosing a cavity—cœlom—between them. The cœlomic cavity is continuous with the extra-embryonic cavity. The external layer of the split mesoderm remains applied to the ectoderm and forms with it the somatopleure. The corresponding internal layer of mesoderm remains applied to a portion of the entoderm of the primitive gut and forms with the entoderm the splanchnopleure. The splanchnopleures from both sides unite in front to complete the intestinal tube. The united splanchonopleures fuse with the wall of the vitello-intestinal duct. Inferiorly, the union of the splanchnopleures closes over the primitive entodermal cloaca.

The alimentary canal extends from the buccopharyngeal membrane to the cloacal membrane; the attachment of the canal varies in its several portions. The foregut is attached to the walls of the entodermal pleuroperitoneal channels. The terminal foregut, midgut, and proximal hindgut are suspended in the body cavity by a continuous vertical mid-dorsal attachment to the angles of union of the split mesoderm of each side. The blood and nerve supply of the gut enters its posterior wall between the dorsal attachments. The dorsal attachments of the gut become attenuated and form the two leaves of its mesentery.

The hindgut caudal to its termination of the mesentery expands in a bay which occupies the interior of the tail fold and empties at its ventrocephalic border in the narrow channel of the allantois. The portion of the bay caudal to the origin of the allantois is the internal cloaca. The cloaca is lined throughout with entoderm.

The lateral walls of the entodermal cloaca are the paired inferior wings of unsplit mesoderm whose peripheral margins form the mesodermic borders of the cloacal membrane. The ventral wall or floor of the cloaca is the cloacal membrane. Dorsal to the cloaca are the axial structures of the notochord and neural tube. The cranial wall or roof is formed by the union of the inferior portions of the splanchnopleures, and is the floor of the cœlomic cavity.

The surface cells of the mesoderm lining the interior of the cœlomic cavity become flattened and form the continuous sheet of mesothelium or peritoneum.

PART II

UROGENITAL APPARATUS AND UROGENITAL FOLD—KIDNEYS AND
BLADDER—GENITAL GLANDS—UROGENITAL UNION, GROWTH
AND DEGENERATION—FINAL CHANGES IN UROGENITAL
FOLD WITH CHANGED POSITION OF GENITAL GLANDS

Urogenital Apparatus, Kidneys and Bladder. The dorsal abdominal wall, between the intestinal mesentery and the lateral parietes is the region of the mesoderm of the intermediate cell mass. Faint transverse grooves mark off the longitudinal area into segments. Beginning above, each segment develops an outgrowth of solid mesoderm dorsally to form the pronephros. The outgrowth extends horizontally dorsad, and approaching the ectoderm turns caudad to unite with the caudal portion of the succeeding segment below. The caudal portion of the outgrowth from the terminal segment fuses with the ventrolateral wall of the cloaca near the cloacal membrane. The horizontal outgrowths disappear as their caudal portions unite. The union of the caudal extensions of the mesodermal downgrowths forms a long cord—primary excretory cord—attached to the wall of the cloaca.

The primary excretory cord runs in the mesoderm at first close to the ectoderm and then bends ventrally towards its attachment on the ventromedial portion of the wall of the cloaca close to the cloacal membrane.

The pronephros does not function as an excretory organ. While the pronephros is disappearing, horizontal outgrowths appear from the same parent nephrogenic tissue, and advance directly into the primary cord at its side. The mesonephros, or Wolffian body is formed. A continuous lumen develops in the horizontal outgrowths and their attached long cord and the secretory tubules of the mesonephros communicates by its primary excretory or Wolffian duct with the entodermal cloaca. The simple horizontal tubules become coiled. The wall of the proximal coil becomes invaginated and a vascular tuft grows against this wall to form a Malpighian corpuscle. The mesonephros functions as the provisional kidney of the fetus.

The Wolffian fold of each side lateral to the excretory duct becomes depressed by a cornet-shaped evagination into the fold
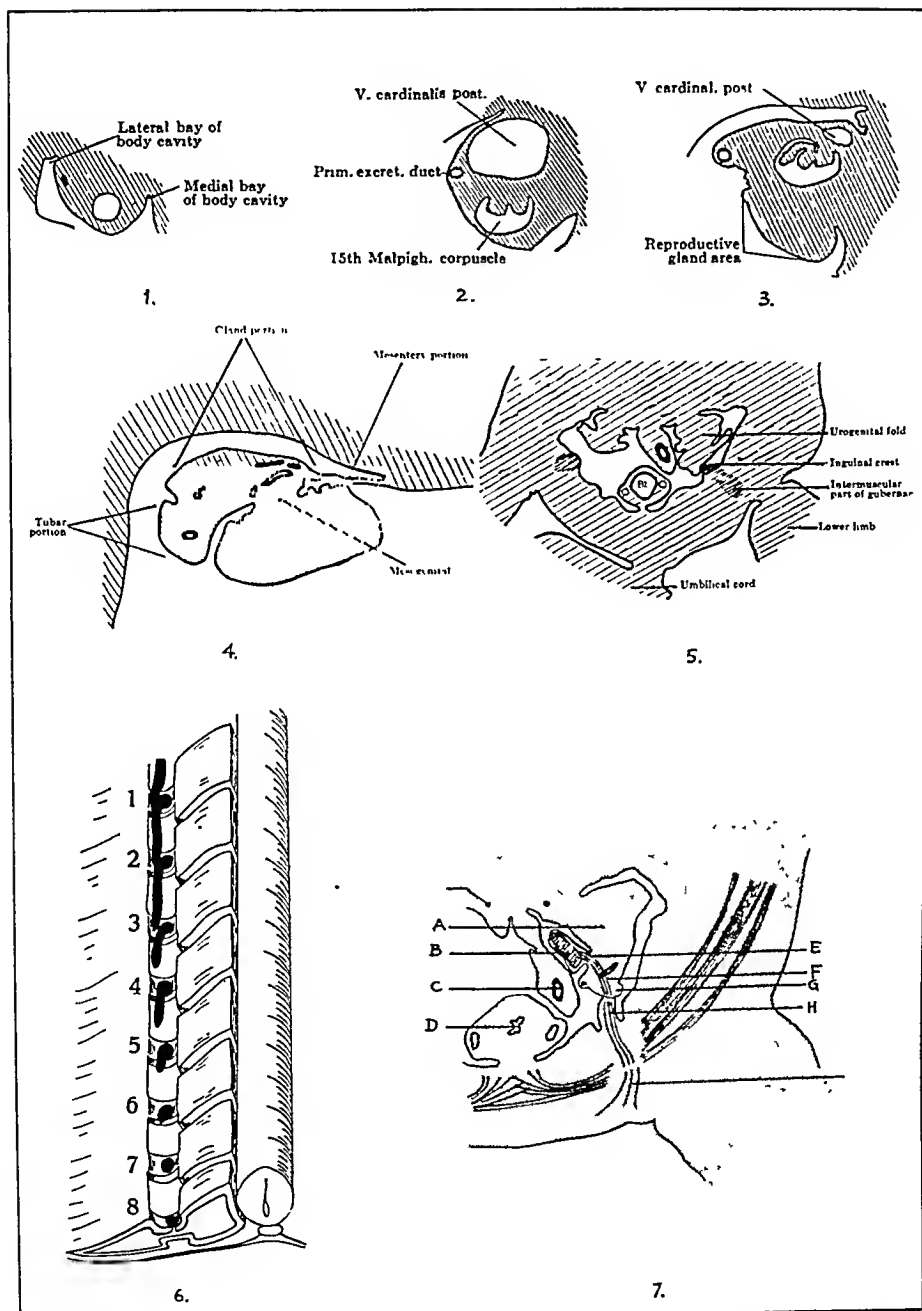
PLATE VIII. 1–5 and 7, transverse sections including body cavity of successive stages of the urogenital fold. 2, embryo of 12.5 mm. (Hochstetter). 6, pronephros—dorsal view showing union of caudal extensions. A, urogenital fold. B, testicle. C, intestine. D, bladder. E, genital ligament. F, inguinal ligament. G, inguinal crest. H, intermuscular part of chorda gubernaculi. Terminal fibers at base of labioscrotal fold as in 5 and 7. (After Felix.)

by a downgrowth of mesothelium from the surface. The downgrowth continues and forms the Müllerian ducts.

The primary excretory duct, before its insertion into the wall of the cloaca, develops a diverticular evagination—ureteric bud—from its dorsolateral wall. The ureteric bud grows dorsocephalad into the surrounding nephrogenic mesoderm. The mesoderm is compressed by the growing bud to form a covering layer for its tip—metanephrogenic cap. The tip of the ureteric bud and its cap of mesoderm form the metanephros or permanent kidney.

The elongating ureter advances the metanephros caudocephalad, deep to the mesonephros. The single ureteral bud undergoes successive divisions which impede the upward course of the kidney. The kidney comes to rest under the enormously growing suprarenal body.

The branching of the ureteral tip divides the metanephric cap into isolated segments. Each segment develops a lumen to form a vesicle. The vesicle elongates into a tubule which becomes convoluted, and, finding by its distal end the extremity of a terminal subdivision of the ureteric tree, unites with it to form a continuous tubular structure that empties into the ventral cloaca. A tuft of vessels follows the invagination of the proximal coil and a Malpighian corpuscle is formed. The permanent kidney is established. The kidney and its ureter are retroperitoneal. Each kidney lies under a portion of the colon and its ureter is crossed by the structures that develop from the urogenital fold.

The lumbodorsal anteflexion of the embryo constricts the middle portion of the urogenital cloaca against the pubis. The cavity of the ventral cloaca expands above as the bladder, narrows at the pubis as the pelvic portion of the cloaca and expands inferiorly as the urogenital sinus. The walls of the ventral cloaca taper above as they pass from the expanded portion into the mesoderm of the umbilical cord that supports the allantoic continuation of the cloacal cavity. The allantois is the principal attachment of the bladder.

The Wolffian duct with its ureteral bud is attached to the bladder. As the bladder expands the ureteric diverticulum is taken up into the expanded wall. The common duct of the ureter and Wolffian duct empties into the cavity. The ureteric orifice in the bladder remains patent. The terminal piece of the excretory duct replaces the related portion of the bladder and the duct opens into the bladder at a lower level and more medially. The bladder, with its laterally placed ureters is carried upward by the cephalad migration of the

umbilical cord. The excretory ducts open into the pelvic portion of the urogenital cloaca on its dorsal surface.

The entodermal wall of the cloaca between the orifices of the ureters and the excretory ducts is replaced by the mesoderm of the excretory ducts. This area of mesoderm is the trigone of the adult bladder.

Urogenital Fold. The development of the mesonephros in the dorsal body wall forms a bulge—urogenital fold—into the cœlomic cavity. The fold is early covered by epithelium spread uniformly in two layers. Later the epithelium on the summit and median slope of the fold to the root of the mesentery becomes many layered, and constitutes the reproductive gland area. This extends throughout the length of the median half of the urogenital fold. Epithelium from the thickened area grows into the fold and compresses its loose mesonephric zone into a strip of denser tissue which becomes sharply defined from its overlying epithelium. The Malpighian corpuscles and mesonephric tubules are displaced dorsally. The growth of the ingrowing epithelium forms a solid epithelial mass— genital gland—with a distinct boundary from the subjacent mesoderm. The epithelial mass separates into a superficial epithelium and epithelial nucleus. When the mass attains a third of the thickness of the urogenital fold the genital gland forms a mound on the fold which becomes marked off in the urogenital fold by a groove at the boundary between the epithelial mass and the Wolffian body that is incomplete above and below. The gonad is free at the sides and continuous with the urogenital fold above, below and by its median portion. In the female, the urogenital union becomes nonfunctioning, but in the male it remains functioning. The portion of the urogenital fold between the fossae of the grooves is the stalk of the genital fold. The outer portion of the mesonephric fold containing the Müllerian tubes is marked off by a groove and the broad attachment of the fold to the dorsal abdominal wall becomes attenuated. The mesonephric fold consists of three portions: the tubar, the common portion of the mesonephric gland and excretory duct, and the dorsal mesentery. The stalk of the genital gland is attached to the mesonephric gland portion of the fold.

General Distinctions of Gonads. The reproductive glands of both sexes develop from the epithelial nucleus of the genital fold. In the male the epithelium forms cords which acquire a connective tissue investment to form the primitive testis. Many of the cords become hollowed out to form the tubules of the testis. The form of the cord and later of the tubule is determined by the indifferent cells from

which they principally originate. The genital cells or spermatogonia have no influence on the form. In the female the epithelial nucleus is passively divided by inwandering connective tissue into a network of masses and cords of cells to form the ovary. Each genital cell forms a unit by itself. The form of the primary follicles is determined by their ova.

The covering layer of the tunica albuginea of the sex glands is formed from the outermost layer of the epithelial nucleus. The portion of the epithelial nucleus that projects into the genital stalk forms the rete cords of the sex gland.

Urogenital Union. The invasion of the urogenital fold by the ingrowing epithelial nucleus of the genital gland is associated with a degeneration of the lower mesonephric secretory tubules as far as their transition into the collecting tubules. The distal ends of the collecting ducts lie in an epithelial bay of the genital rete cords from which they are sharply defined. The cords become tubular and, uniting with the distal ends of the collecting tubules, effect the urogenital union. The collecting ducts are still connected with their excretory—Wolffian—duct.

Male Sex—Changes in the Fold; Development and Degeneration. In the male the collecting ducts that have united with the rete tubules form the efferent ducts of the testis. The testicular piece of duct remains straight while the portion attached to the Wolffian duct becomes coiled. The straight portion is surrounded by firm connective tissue and becomes the comus vasculosi. The coiled portion includes the lower end of the excretory duct to become the canal of the epididymis. The remainder of the Wolffian duct becomes the vas deferens. The vas deferens dilates to form an ampulla from which the seminal vesicle develops. Its narrow horizontal portion, the ejaculatory duct opens into the urethra. The epithelium of the duct is ciliated.

Degeneration of the Fold. The Müllerian ducts in the male begin to degenerate shortly after their union to form the uterovaginal canal on the urogenital cloaca. The portion of the tube between the caudal pole of the testis and the uterovaginal canal breaks down, leaving a very small portion situated between the head of the epididymis and the testis—the hydatid of Morgagni. This remnant usually contains a simple epithelial canal. The uterovaginal canal persists in the broad horizontal portion—vagina masculina. Müller's tubercle persists as the colliculus seminalis. The cristae urethralis are early defined.
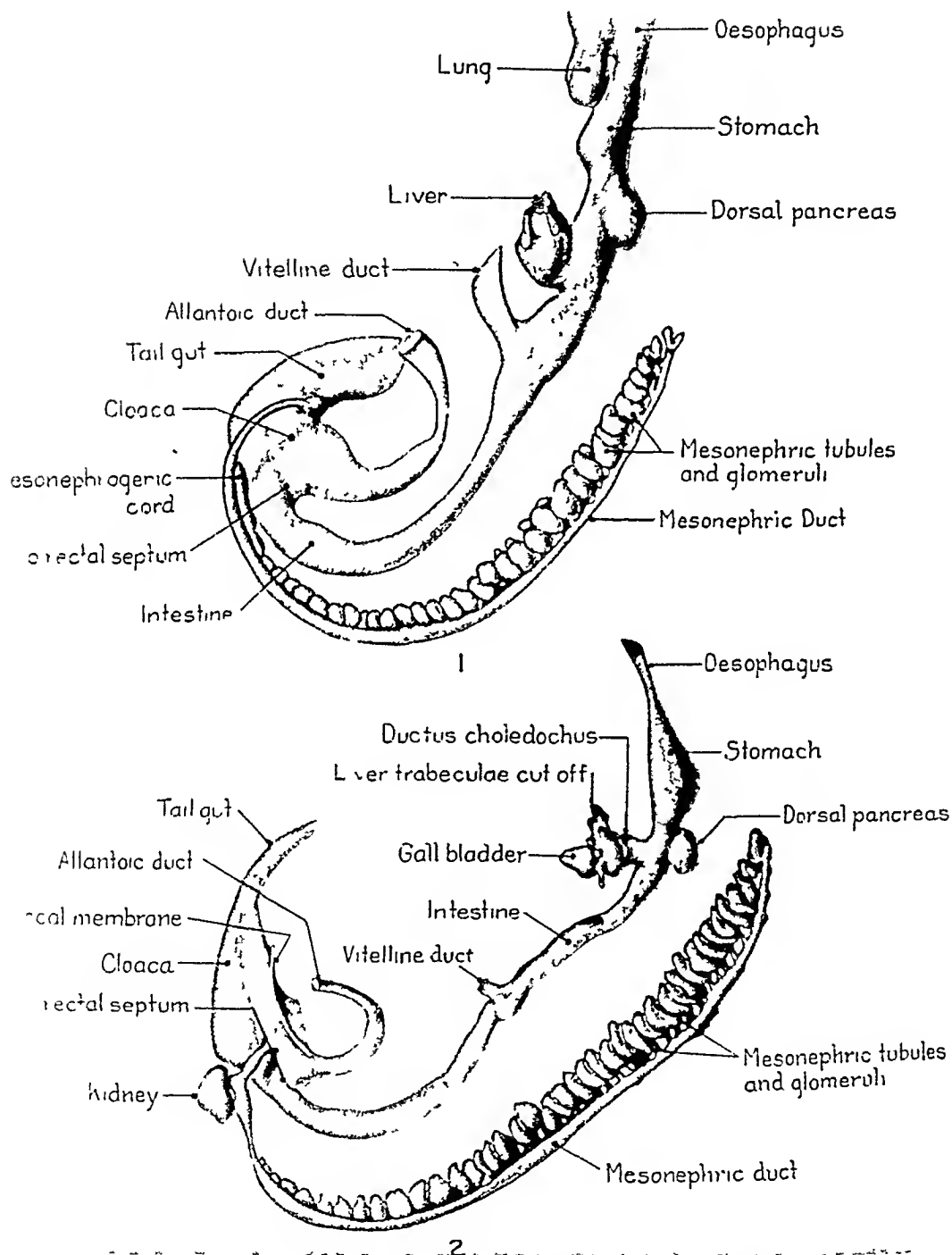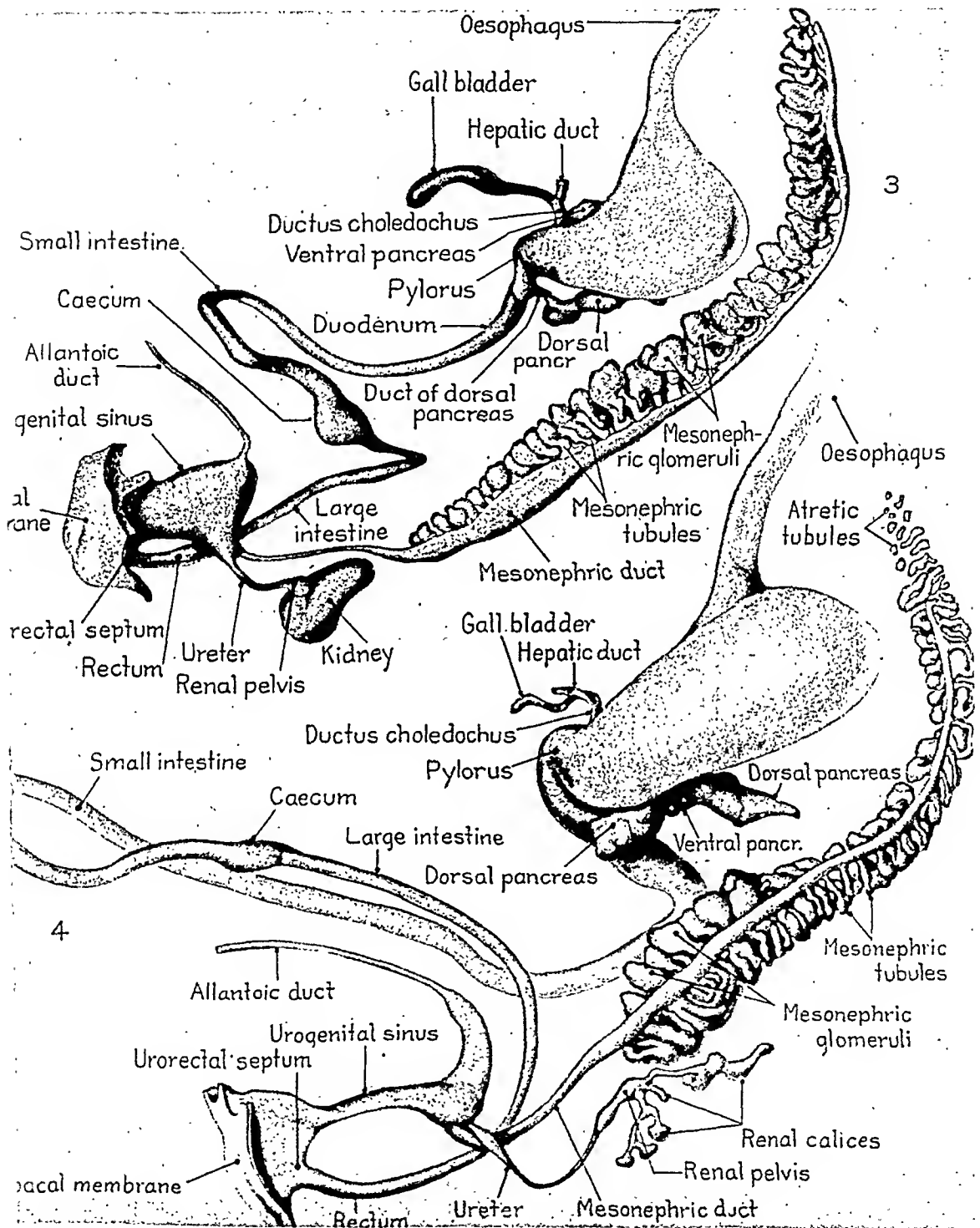
Oesophagus

Lung

Stomach

Liver

Dorsal pancreas

Vitelline duct

Allantoic duct

Tail gut

Mesonephric tubules
and glomeruli

Cloaca

Mesonephric Duct

mesonephiogenic
cord

 rectal septum

Intestine

1

Oesophagus

Ductus choledochus

Liver trabeculae cut off

Stomach

Tail gut

Dorsal pancreas

Allantoic duct

Gall bladder

cal membrane

Intestine

Cloaca

Vitelline duct

rectal septum

Mesonephric tubules
and glomeruli

kidney

Mesonephric duct

2

PLATE IX. 1, mesonephros, alimentary canal in 4 mm. human embryo. 2, mesonephros, metanephros
embryo. 4, same, showing further development

Oesophagus

Gall bladder

Hepatic duct

Ductus choledochus

Ventral pancreas

Pylorus

Duodenum

Dorsal pancr

Small intestine

Caecum

Allantoic duct

genital sinus

al rane

Duct of dorsal pancreas

Large intestine

3

Mesonephric glomeruli

Mesonephric tubules

Mesonephric duct

Oesophagus

Atretic tubules

rectal septum

Rectum

Ureter

Renal pelvis

Kidney

Gall bladder

Hepatic duct

Ductus choledochus

Pylorus

Dorsal pancreas

Ventral pancr.

Mesonephric tubules

Small intestine

Caecum

Large intestine

Dorsal pancreas

Mesonephric glomeruli

4

Allantoic duct

Urogenital sinus

Urorectal septum

Renal calices

Renal pelvis

acal membrane

Rectum

Ureter

Mesonephric duct

and alimentary canal of 5.5 mm. human embryo. 3, same as 2, showing further development in 8 mm. in 14.6 mm. embryo. (After Shikinami.)

Mesonephros. Degeneration of the mesonephros begins with the completion of its cranial growth. Proceeding from above down the tubules of the cervical and thoracic segments disappear with their corresponding portion of the Wolffian duct. Later, the remaining tubules, all in the lumbar region are in some stage of degeneration. The upper tubules unite with the sex gland, and the Wolffian remnant consists of an upper genital portion or epigenitalis, and a lower mesonephric gland portion, the paragenitalis. The break in the tubules occurs at the junction of the collecting ducts with the secretory tubules. The mesonephric tubules attached to the Wolffian duct lie in the urogenital fold that has gained attachment to the anterior abdominal wall. The urogenital fold follows the wall and, becoming drawn out, forms a double bend to enter the true pelvis. The Wolffian duct is carried by the fold, and in its horizontal portion receives the paragenitalis. The excretory duct forms a loop that carries its attached collecting ducts away from the secretory tubules and Malpighian corpuscles. The latter structures remain caudal to the epigenitalis and may be present in cranial and caudal groups. The first group of corpuscles and tubules may persist, but usually disappears. In the latter case there is one paragenitalis; in the former there are two.

The collecting tubules carried away with the Wolffian duct may have their individual orifices in the duct reduced to a single orifice which forms the collecting duct of the paradidymis or the canaliculus aberrans Halleri. The paragenitalis forms the paradidymis, or the organ of Giraldis. When present it lies between the head of the epididymis and the testis.

Changes in position of the testis and its duct are influenced by related structures. The testicle as a member of the urogenital fold retains its attachment to the fold and its position is an incident in the changing position and attachments of the fold. As a projection into the body cavity the testis is associated with the peritoneal cavity of its immediate vicinity.

The urogenital fold begins as a bulge from the dorsal body wall that extends longitudinally from the diaphragm to the dorsal wall of the ventral cloaca. The fold at this time is attached by a broad base to the dorsal wall. The lateral wall is narrow, and the anterior wall is non-existent.

As the urogenital structures become defined in the corresponding subdivisions of the fold, the broad base of the fold is defined from its dorsal wall as a stalk which becomes attenuated to form the mesentery of the fold. The mesentery attached by its upper portion

Atretic tubules

Renal calices

Müllerian duct

Oesophagus

Mesonephric tubules and glomeruli

Renal parenchyma

Dorsal pancreas

Hepatic duct

Ductus choledochus

Mesonephric duct

Cystic duct

Ureter

Pylorus

Duodenum

Gall bladder

Rectum

Urachus

Uro rectal septum

Small intestine

Large intestine

Urogenital sinus

Anus

Caecum

Vermiform appendix

5
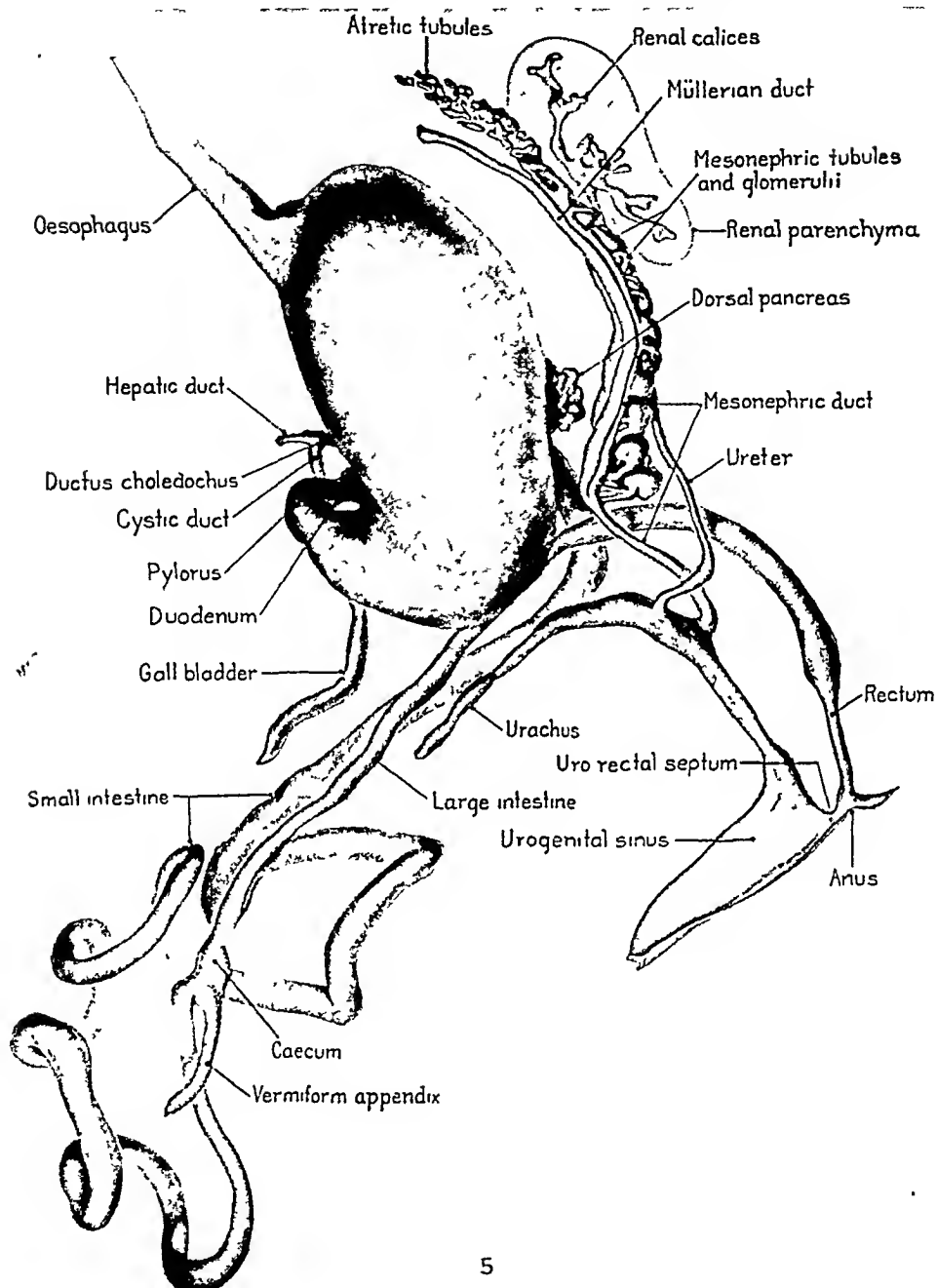
J. F. Didusch fecit

PLATE X. Continuation of Plate IX. 5, same as 4, showing development in 23 mm. embryo. Urorectal septum is the pelvic bar. (After Shikinami.)

to the diaphragm is the diaphragmatic ligament of the mesonephros. The mesenteric ligament keeps pace with the degenerating mesonephros as it becomes reduced from above down and takes progressively lower attachments on the dorsal wall until the spermatic vessels, growing into it, convert the diaphragmatic ligament of the mesonephros into the permanent mesentery of the spermatic vessels. By this time, the mesonephros has collapsed and the testis through its mesorchium has acquired the diaphragmatic ligament of the Wolffian body.

In the beginning the urogenital fold of each side lies parallel to the vertebral column. Organs developing in the midline force the upper portions of the fold away from each other. The enormous growth of the suprarenal bodies and the metanephroi in the dorsal wall spread the folds laterally. The large stomach and intestinal coils further displace the folds towards a horizontal position. Room for the displacement has been provided by the broadening of the dorsal wall. Below the metanephroi, the folds retain their sagittal position. The fold forms a double bend as it lies between its displaced upper and undisturbed lower portions. The bent urogenital fold consists of three portions; an upper sagittal, a middle horizontal, and a lower sagittal. The lower sagittal portions unite with the lower dorsal wall of the ventral cloaca and with each other. The area of fusion is the genital cord and contains the fused Müllerian tubes and the primary excretory ducts. The Müllerian ducts disappear as far as their fused portion which persists as the vagina masculinus. The horizontal portion of the bent fold develops a fold at its outer bend—inguinal fold—which grows towards the dorsum of the lateral wall to unite there with the ridge—inguinal crest—formed in the lateral wall. The horizontal urogenital fold, comprising the medial genital and lateral mesonephros folds, is attached to the lateral wall by the inguinal ligament of the mesonephric fold, and accompanies the wall as it is pushed anteriorly.

The genital fold and its contained gland change their form by cranial degeneration and caudal growth. The gland occupies the fold between its ends. The lower end of the fold—ligament testis— attaches the gland to the lower sagittal portion of the urogenital fold that enters into the genital cord. The caudal pole of the gland, which lies above the true pelvis, becomes constricted off from the mesonephric fold. The ligament of the testis at the junction of the true and false pelvis is attached to the medial portion of the horizontal urogenital fold which is attached to the inguinal crest of the lateral body wall by the inguinal fold.

The inguinal crest, before the mesoderm of the wall has differentiated into muscle, develops a compact cord—chorda gubernaculi—which extends through the thickness of the mesodermal body wall from its apex at the crest to its base near the integument. The musculature of the abdominal wall, as it becomes differentiated from the mesenchyme, grows around the persisting chorda gubernaculi. The cord lies in a canal of the musculature of the abdominal wall. The transversus and the internal oblique provide a mantle of fibers that extend up on the cord to the inguinal crest. The external oblique forms a foramen at which the cord ends by its base. From the foramen a cord of mesoderm forms—ligamentum scroti—that extends to the deep surface of the labioscrotal folds on the perineum. The aponeurosis of the external oblique grows around the ligamentum scroti and forms the cremasteric fascia. The cords of the ligamentum testis, inguinal folds, chorda gubernaculi, and ligamentum scroti unite to form a continuous cord that extends from the caudal pole of the testis, through the inguinal canal to the scrotal sac.

The caudal pole of the testis, the attached mesonephric fold and the gubernaculum at first project into the center of the portion of the abdominal cavity cut off from the main space—saccus vaginalis—and are covered with peritoneum both medially and laterally. Later, the saccus covers only the medial protruding portion when the lateral surfaces of the urogenital fold fuse with the lateral wall of the saccus. Thickening of the anterior abdominal wall fails at the point where the gubernaculum traverses the wall and the developing musculature grows around a small portion of the saccus. The saccus penetrates the musculature and enters the scrotal sac. A narrow peritoneal canal connects the saccus with the abdominal cavity. Shortly after birth the canal becomes solid. The saccus, completely cut off from the abdominal wall, becomes the tunica vaginalis propria of the testis which has wandered through the canal into the scrotal sac.

Female—Degeneration of the Urogenital Union. Changes in the structures of the urogenital folds of the differentiated ovary correspond in general to the changes that take place in the male, with the important differences that the excretory duct disappears in the female and the growing uterus intercepts the caudal ligament of the genital gland.

The mesonephros degenerates in its cranial portion and consists of the upper epigenitalis—epoöphoron—attached to the rete ovarii and the lower paragenitalis—paroöphoron—of the remaining

mesonephric gland. The paroöphoron quickly degenerates in extra-uterine life.

The primary excretory duct, the functionless duct of the ovary, degenerates before the rete ovarii has united with the mesonephros. The epoöphoron is free when it develops. The degeneration of the Wolffian duct takes place at the same time that the Müllerian duct disappears in the male. The excretory duct generates in its portion between the caudal pole of the ovary and the beginning uterovaginal canal. The remains of the excretory duct constitute Gartner's canal. The canal may retain connection with the epoöphoron for a time. The canal first lies in the broad ligament, then sinks into the musculature of the uterus at the internal os, runs through the musculature to pass through the portio vaginalis into the lateral fornix of the vagina. The lower portion passes through the wall of the vagina and, passing through the vestibular epithelium, opens to the exterior near the free border of the hymen. Portions of the canal are present in one fourth of females. The persisting Gartner's canal may form an ampulla and a seminal vesicle in the upper part of the portio vaginalis.

Changes in position of the ovaries and their ducts are influenced by related structures. The ovary, as a member of the urogenital fold, retains its attachments to the fold and its final position is an incident in the changing position and attachments of the fold.

The urogenital fold begins as a bulge of the dorsal body wall into the peritoneal cavity and extends longitudinally from the diaphragm to the dorsal wall of the ventral cloaca. The fold at this time is attached by a broad base to the dorsal wall. The lateral wall is narrow and is yet to form the anterior wall. The structures in the urogenital fold occupy corresponding divisions of the subdivided fold which are connected to each other. The broad base of the urogenital fold at its cranial end becomes a thready mesentery, the diaphragmatic ligament of the mesonephric fold. The ovary occupies the genital fold, and the remainder of the urogenital fold is divided into the tubar portion for the Müllerian tubes, the gland portion for the mesonephros and its excretory duct, and the diaphragmatic ligament of the mesentery. The mesonephric fold joining the tube and ovary to the diaphragmatic ligament degenerates and the developed ovary and its tube acquire the mesenteric portion of the urogenital fold. The mesentery forms divisions for the tube, infundibulopelvic ligament, and for the ovary, the ovaricopelvic ligament. The ligaments, thickened with the ingrowth of blood vessels,

retain their connection with the degenerated mesonephric gland portion of the urogenital fold.

The urogenital folds, at first parallel to the vertebral columns, are displaced laterally above the true pelvis by the medial supra-renals and metanephroi of the dorsal body wall and the stomach and intestines of the abdominal cavity. Each fold forms a double bend between the upper sagittal and lower sagittal portions. Between the two bends is the horizontal portion.

The lower sagittal urogenital folds unite first with each other in the floor of the pelvis to form the ventral partition of the cavity, and then with the inferior dorsal wall of the ventral cloaca. The union of the folds forms the genital cord. The genital cord divides the pelvic cavity into the recto-uterine and vesico-uterine fossae. The upper end of the genital cord lies at the level of the second bend of the fold.

Within the genital cord, the Müllerian ducts lie between the excretory ducts and the whole mass forms a projection of the dorsal wall of the vesico-urethral sinus to form the Müllerian tubercle on its interior. The Müllerian ducts grow to the tubercle and then run horizontally on the wall of the sinus. The horizontal portion of the ducts is solid. The upward growth of the vesicle portion of the ventral cloaca leaves the Müllerian tubercle as a projection on the dorsal wall below it.

The right and left Müllerian tubes unite by their median external surfaces within the genital cord and form the paired uterovaginal canal. The attached median walls fuse to form a septum which becomes absorbed in the caudocranial direction to form the single uterovaginal canal. The Müllerian tubes, at their abdominal origin of a single funnel, acquire secondary funnels. The tubes on their completion of the uterovaginal canal run vertically downwards in the lateral tubar portion of the mesonephric fold, to the edge of the genital cord, bend there across the excretory ducts to run horizontally towards the midline to unite in the uterovaginal canal. Each Müllerian tube consists of a vertical, a horizontal and a fused portion in the uterovaginal canal. The vertical portion becomes the tube of the adult; the horizontal portion forms its half of the fundus of the uterus; and the fused portion the cervix and upper four fifths of the vagina. Interference with the union of the two tubes by the ventral structures of the body wall results in various anomalies of the uterovaginal canal.

The horizontal piece of the tube lies in the tubal portion of the urogenital fold at the level where the fold develops its inguinal
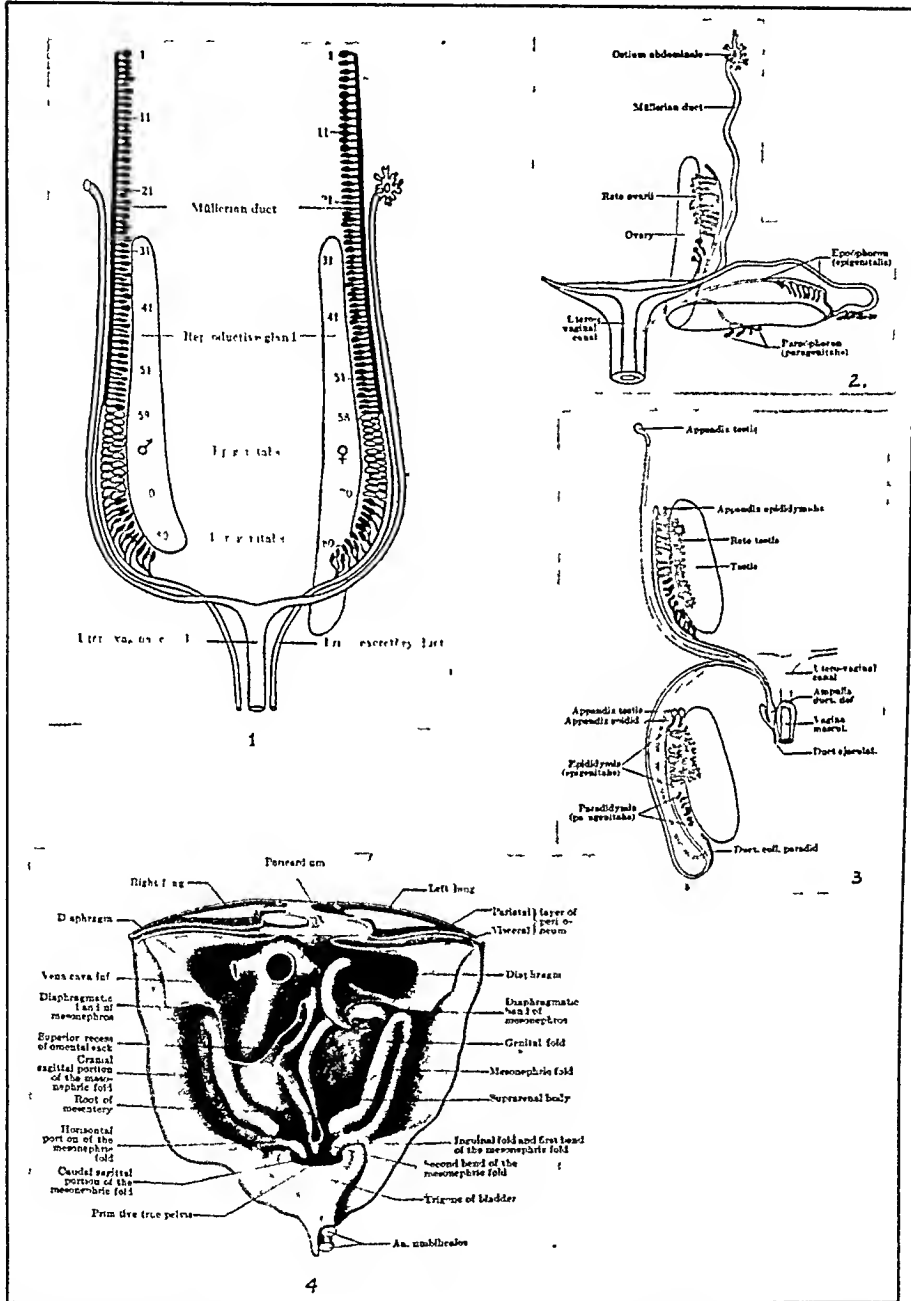
PLATE XI. Urogenital fold and ultimate disposition of its structures. 1, diagrammatie representation of the entire mesonephric organ, reproductive gland, and efferent ducts. Left is male and right is female reproductive gland. 2, after migration of the ovary. Degenerated structures in dotted lines. 3, after migration of the testis. Degenerated structures in dotted lines. 4, model of posterior wall of human embryo of 19.4 mm. (After Felix.)

ligament that attaches to the gubernaculum of the abdominal wall.

The genital fold and its ovary change their form and position by cranial degeneration and caudal growth. The caudal pole of the ovary reaches the true pelvis behind the genital cord of the united mesonephric folds. The ovary occupies the fold between its ends. The lower end of the genital fold—ligament of the ovary—attaches the caudal pole of the ovary to the genital cord. The pathway of the ovarian ligament through the horizontally bent urogenital fold to the inguinal ligament for the abdominal wall is interrupted by the growth of the uterus from the horizontal portion of the Müllerian tube. The ovarian and inguinal ligaments growing to each other are intercepted by the uterus to which each gains an attachment. The inguinal ligament from its subserous attachment to the uterus unites with the chorda gubernaculi of the abdominal wall—round ligament of the uterus—which continues as the ligamentum labiale to the deep surface of the integument of the labial fold of the external surface. The inguinal canal and the peritoneal extension into the canal, form as in the male.

The ovary rotates around the caudal pole through an angle of 90 degrees and its cranial pole takes a lateral position. The tube portion of the Müllerian duct is carried laterally by the rotation of its attachment to the dorsal abdominal wall, the attenuated base of the original urogenital fold, and takes a lateral position above the ovary. The cranial portion of the tube is in relation to the cranial portion of the ovary during development and after rotation.

The urogenital sinus, at each side of its posterior wall, develops evaginations—sinovaginal bulbs—which extend to the Müllerian tubercle and fuse with it. The bulbs become solid by the proliferation of their lining epithelium and fuse with the tip of the Müllerian— uterovaginal—ducts which become solid by the same process. The solid mass of cells is the vaginal plate which grows in all directions. The central cells of the solid vagina break down to form the cavity of the vagina. The cranial end of the vagina is defined by the formation of the anterior and posterior fornices as solid epithelial projections. The caudal end expands and pushes in the posterior wall of the urogenital sinus and extends along the posterior wall to form the caudal segment of the hymen. The anterior paired segment of the hymen is formed where the evaginations of the urogenital sinus occur to form the sinovaginal bulbs. The vagina is formed in its lower one fifth from the sinovaginal bulbs which arise from the epithelium of the urogenital sinus.

PART III

ENTODERMAL CLOACA—DIVISION PELVIS, PELVIC BAR, UTEROVAGINAL CANAL, BLADDER, URETHRA, GLANDS OF UROGENITAL SINUS

Entodermal Cloaca. The cloacal membrane, which provides the sites of the orifices on the surface of the caudal end of the body, the future perineum, appears before the primitive streak is completed. At this time the membrane is the small area of the caudal surface of the embryonic disc where the ectoderm and entoderm are most intimately apposed.

The ectodermal amniotic sac grows posteriorly against the body stalk and eats its way through the morula mesoderm of the stalk to the entodermal lining of the allantois. The area of ectoderm closely applied to entoderm has been extended from the cloacal membrane of the embryonic plate for a short distance on the dorsum of the body stalk. The mesoderm of the embryonic plate condenses into sheets with superior and inferior wings. The inferior wings of mesoderm run along the lateral borders of the extended cloacal membrane and lose themselves in the morula mesoderm of the body stalk. The axial structures grow down to the upper angle of the membrane. The tail fold forms, and the cloacal membrane extends ventrally from the posterior termination of the cloaca to the body stalk. At this stage the membrane lies in the first plane of the perineum. The lateral borders of the membrane are the medial margins of the inferior wings of mesoderm. The cavity enclosed by the tail fold is the entodermal cloaca which empties in the allantois of the stalk. The inferior wings compose the lateral walls of the cloaca and, when the mesoderm splits to form the body cavity and the intestinal tube, remain unsplit. The cloaca becomes closed off from the abdominal cavity by the union of the lowermost splanchnopleure. The cloaca is the expansion of the hind gut, and terminates superoventrally in the allantois of the stalk. The partition of the fused inferior splanchnopleures separates the cavity of the cloaca from the abdominal cavity. The ventral wall of the cloaca is the cloacal membrane. The cloaca occupies the future pelvis. The partition between the cloaca and the abdominal cavity sags along the ventrolateral borders of the hindgut. The abdominal cavity is extended inferiorly to form the pelvic cavity by the sagging partition.

The caudal portion of the embryo grows beyond its junction with the cloacal membrane to form the tail. The tail carries out a portion

of the cloacal cavity as the tail gut, which soon disappears. The tail curves ventrally above the cloacal membrane toward the body stalk and the perineum is carried forward to face cranially. The tail curves backwards on itself and gradually degenerates. The recurving tail draws the perineal plane forward and downward. At this time the lowest point of the sagging partition has arrived by its entodermal layer at the entodermal layer of the cloacal membrane, and the two layers fuse. The partition is splanchnopleure composed of mesoderm lined on its abdominal surface by peritoneum, and on its cloacal surface by entoderm.

The migration downwards of the splanchnopleure forms the extension inferiorly—pelvic cavity—of the peritoneal abdominal cavity. The attachment of the partition to the deep surface of the cloacal membrane divides the cloacal cavity into two separate portions, the large ventral urogenital cloaca, and the smaller dorsal rectal cloaca. The rectal cloaca is continuous above with the hindgut. The urogenital cloaca receives the Wolffian duct with its ureteric diverticulum, and terminates superoventrally in its allantoic diverticulum in the body stalk. The posterior wall of the ventral cloaca and the anterior wall of the dorsal cloaca are formed from the splanchnopleure and are lined with peritoneum. These walls are joined to each other by the portion of splanchnopleure that has become attached to the cloacal membrane and forms the urorectal septum or pelvic bar. The pelvic bar is the inferior wall of the pelvic cavity, and separates the cavity from the cloacal membrane of the body surface.

The lateral walls of the cloacal divisions are the inferior wings of mesoderm that have remained unsplit, and their medial margins are the mesodermal borders of the cloacal membrane. The cloacal membrane is the ventral wall of the ventral and dorsal cloacae.

The ventral cloaca is constricted at its middle portion by the forward bending of the caudal embryo. The urogenital cloaca forms

PLATES XII, XIII and XIV show the development of the cloaca. Pelvic structures developed from internal (entodermal) cloaca in relation to perineal structures developed from the external (ectodermal) cloaca. The spaces formed as a result of the development of the pelvic structures are separated from the exterior by various portions of the perineum. (1) The prevesical space, between the bladder and the abdominal wall and pubic bone, is separated from the exterior by the tissue enclosing the dorsal vein of the clitoris or the penis. (2) The prerectal space is separated by the contiguous pelvic and perineal bars. (3) The retrorectal space is separated by the contiguous attachments of the rectococcygeus, levator and external sphincter ani muscles. (4) In the female, the vesicovaginal space is separated by the common distal wall of the urethra and the vagina. The prerectal space is similar to the male. These relations are significant in the surgical anatomy of the pelvis and perineum.
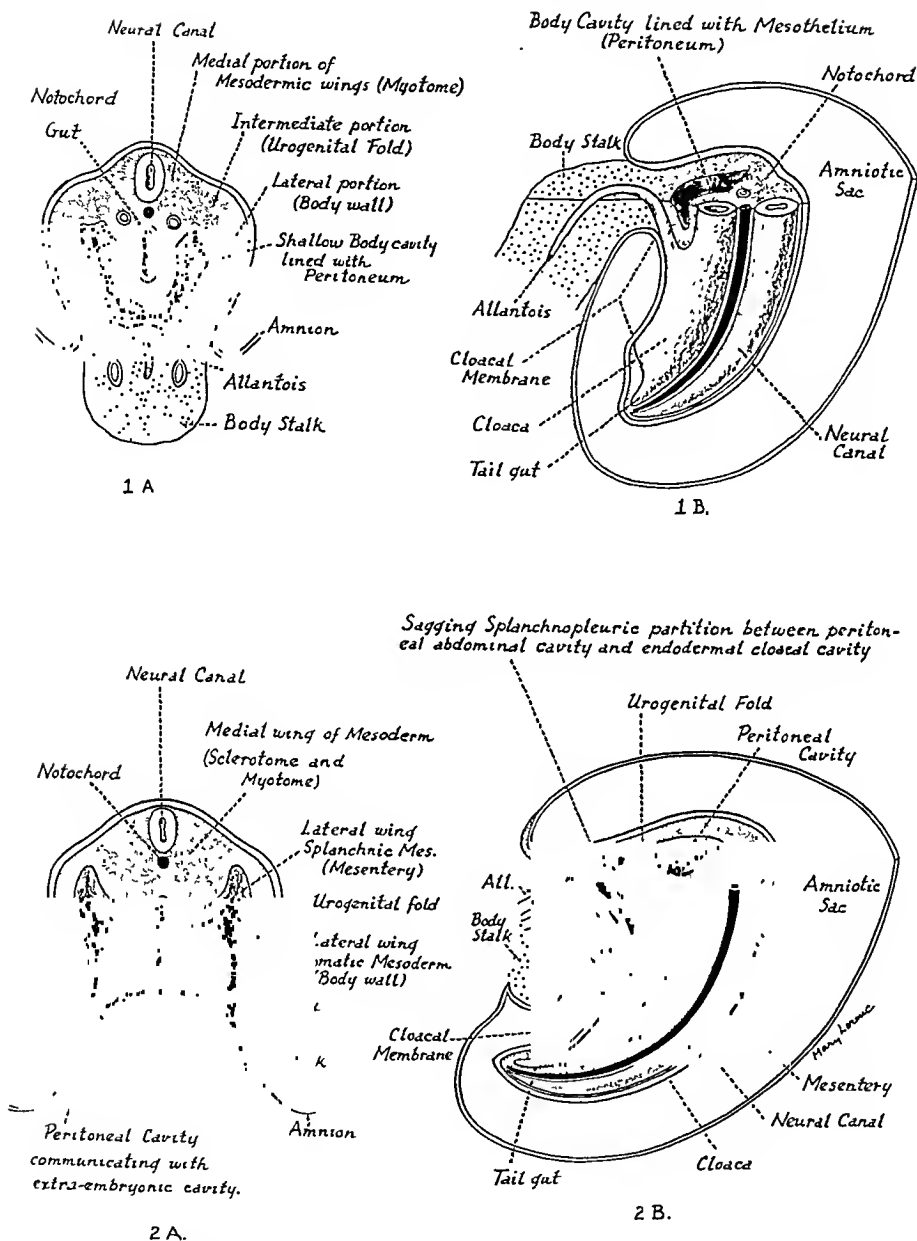
1 A

1 B.

2 A.

2 B.

PLATE XII. Three dimensional sections of embryo in region of cloaca. IA and 2A, cross sections of entodermal cloaca and gut-surrounding mesoderm. Shows peritoneal surface of entodermal cloaca. Shallow peritoneal cavity. IB and 2B, sagittal sections, entodermal cloaca being divided into urogenital cloaca and rectal cloaca by sagging splanchnopleuric partition.
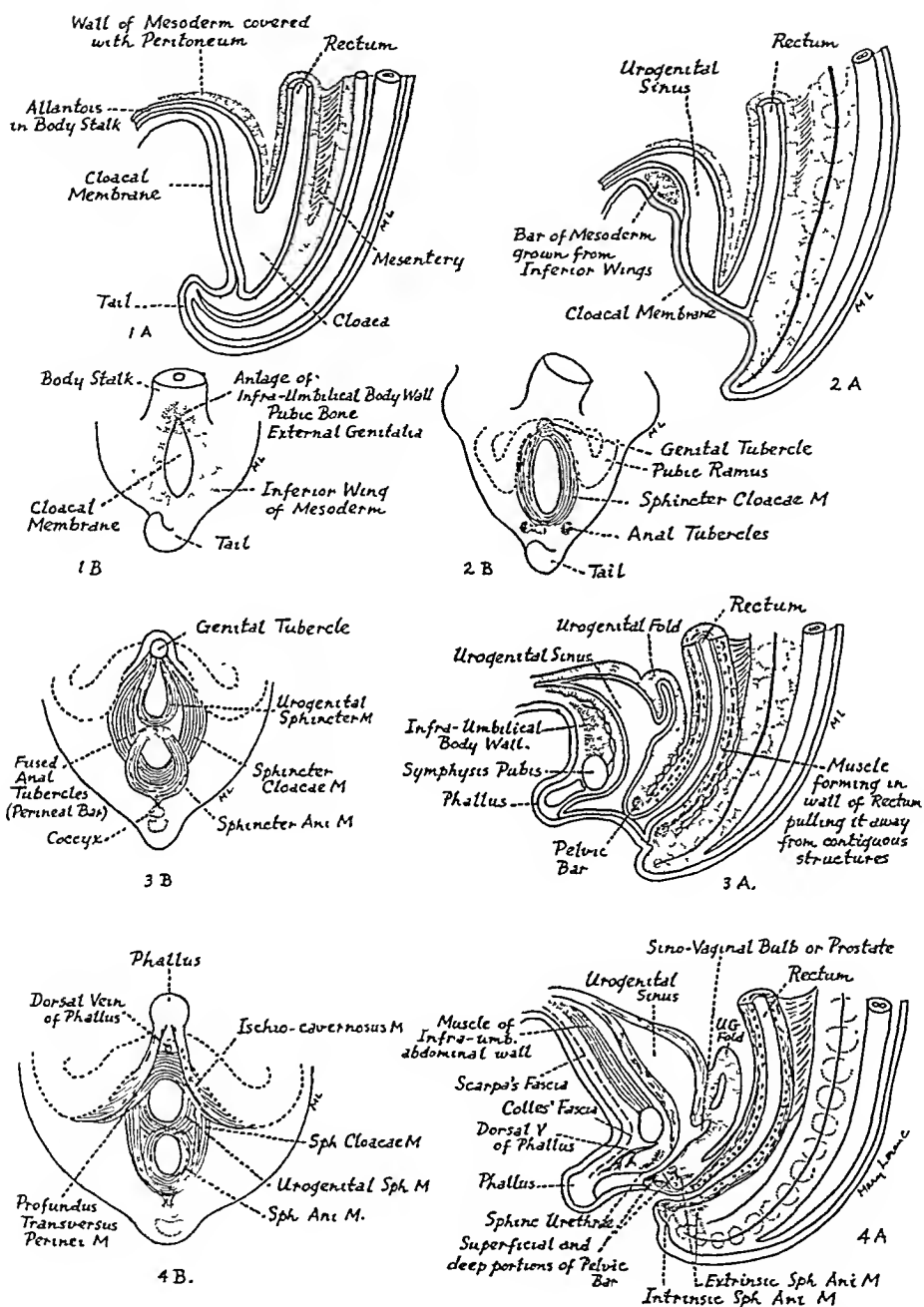
Wall of Mesoderm covered with Peritoneum
Rectum
Allantois in Body Stalk
Cloacal Membrane
Mesentery
Tail
Cloaca
1 A

Urogenital Sinus
Rectum
Bar of Mesoderm grown from Inferior Wings
Cloacal Membrane
2 A

Body Stalk
Antage of Infra-Umbilical Body Wall
Pubic Bone
External Genitalia
Cloacal Membrane
Inferior Wing of Mesoderm
Tail
1 B

Genital Tubercle
Pubic Ramus
Sphincter Cloacae M
Anal Tubercles
Tail
2 B

Genital Tubercle
Urogenital Sphincter M
Fused Anal Tubercles (Perineal Bar)
Sphincter Cloacae M
Sphincter Ani M
Coccyx
3 B

Rectum
Urogenital Fold
Urogenital Sinus
Infra-Umbilical Body Wall
Symphysis Pubis
Phallus
Pelvic Bar
Muscle forming in wall of Rectum pulling it away from contiguous structures
3 A.

Phallus
Dorsal Vein of Phallus
Ischio-cavernosus M
Muscle of Infra-umb. abdominal wall
Sph Cloacae M
Urogenital Sph. M
Sph Ani M.
Profundus Transversus Perinei M
4 B.

Sino-Vaginal Bulb or Prostate
Rectum
Urogenital Sinus
UG Fold
Scarpa's Fascia
Colles' Fascia
Dorsal V of Phallus
Phallus
Sphinc Urethra
Superficial and deep portions of Pelvic Bar
Extrinsic Sph Ani M
Intrinsic Sph Ani M
4 A

PLATE XIII. Synchronization of entodermal cloacal development and differentiation of mesodermal borders of cloacal membrane. A1–4, sagittal sections of caudal portion of body. B1–4, front views of cloacal membrane showing inferior wings of mesoderm and structures developing from them. 1B, distal extremities of inferior wings of mesoderm forming band at upper extremity of cloacal membrane where it extended on the body stalk. 4A, UG fold is the genital portion of the urogenital fold where the uterovaginal canal has formed.
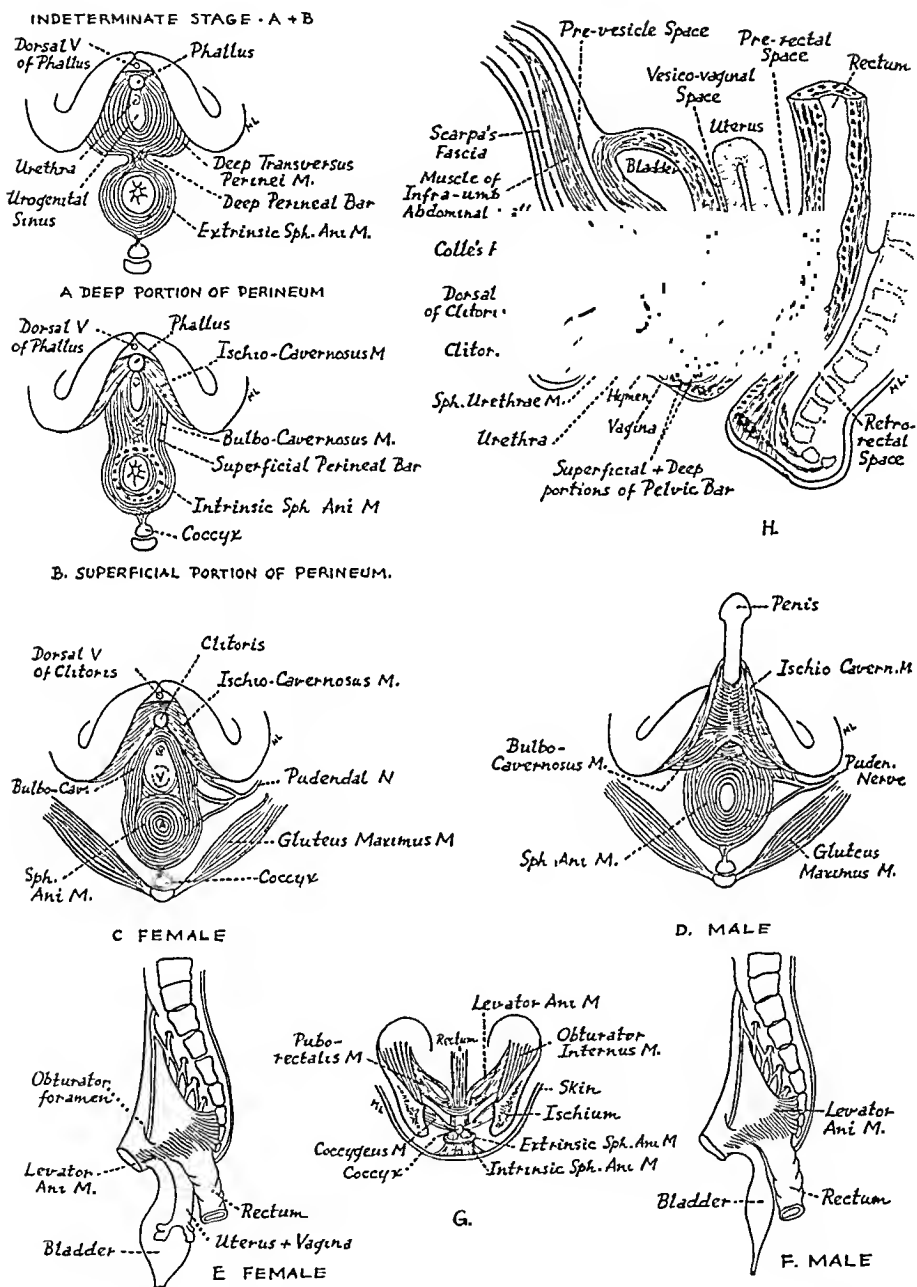
PLATE XIV. Completed pelvic and perineal structures. A and B, indeterminate stage—
perineal musculature, deep and superficial portions, front view. C and D, perineal
musculature, female and male, front views. E and F, sagittal sections, female and male
pelves. G, longitudinal muscle of rectum in relation to levator muscle, external sphinc-
ter ani and skin, showing extrinsic and intrinsic levator and sphincter ani. H, rela-
tions of the peritoneal lining of the pelvic cavity. Retroperitoneal cavities of the
pelvis. Structures separating cavities from the exterior.

an expanded upper portion, the bladder, connected to the allantois; a narrow middle portion, the pelvic urethra; and an expanded inferior portion, the urogenital sinus. The sinus is separated from the exterior by the cloacal membrane. The bladder wall takes in the ureteral orifices and carries them away from the orifices of the excretory ducts as its attached allantois is carried upwards by the cephalad advances of the umbilical cord. The excretory ducts open into the pelvic urethra.

The uterovaginal canal built in the dorsal wall of the pelvic urethra presses its peritoneal wall against the anterior peritoneal wall of the inferior portion of the rectum. The peritoneal walls become applied to each other, and the recess is reduced to a potential cavity. The peritoneum of the reduced pelvic fossa spreads over its gap leading into the potential space and closes it over. The space extends from the pelvic peritoneum above to the pelvic bar below and lies between the rectum and vagina in the female; in the male between the rectum and prostate. The growth of the prostate posteriorly or the growth of the vagina posteriorly, compresses the anterior wall to the posterior walls of the space, and they fuse to form a thickened sheet—Denonvillier's fascia or prostatoperitoneal aponeurosis or vaginal peritoneal aponeurosis, respectively. The active muscular wall of the rectum frees itself from the peritoneal aponeurosis which remains applied to the vagina or prostate. The rectum forms a space —prerectal space—with the peritoneal aponeurosis in front, anterior rectal wall behind, pelvic peritoneal floor above, pelvic bar below. The pelvic bar separates the prerectal space from the cloacal membrane of the external surface. The peritoneal lining separates the pelvic cavity from the prerectal space.

In the female pelvis the uterovaginal canal forms the uterovesical fossa and the uterorectal fossa. The upper four fifths of the vagina is in relation with the dorsal wall of the bladder and proximal urethra.

The urogenital sinus forms the lower fifth of the vagina by evaginations of its posterior walls. The anterior wall of this portion of the vagina is continuous with the dorsal wall of the urethra. Above the level of this attachment the anterior wall of the vagina is apposed to the bladder. Their walls do not fuse. The relation of the vagina to the bladder is similar to the arrangement with the rectum.

GLANDS IN THE UROGENITAL SINUS

The bladder, together with the accompanying umbilical arteries, lies completely within the anterior abdominal body wall. The blad-

der, by expanding and elongating, and by the increased size of the umbilical arteries, is crowded out of the abdominal wall; it develops a ventral mesentery and projects into the body cavity. The two umbilical arteries bound a triangular area on the bladder, vesical plate, whose apex is formed by the umbilicus. Within this area the bladder reaches the umbilicus by its cranial apex with which it connects the remains of the allantois; the internal orifice of the urethra lies at the level of the symphysis. The apex of the bladder diminishes in diameter and finally becomes a narrow tubular extension—urachus—of the bladder. Usually the urachus loses its lumen and degenerates, and is reduced to an epithelial cord. The epithelial cord becomes divided into separate portions which may vanish completely before birth. Persistent remains of the urachus may enlarge in a vesicular manner and form urachal cysts. While the epithelial cord disappears, the surrounding connective tissue persists as a cord and forms the attachment of the bladder—medial vesico-umbilical ligament—of the adult.

The bladder, while it lies in the triangular area between the two umbilical arteries, retains its mesentery; it loses its mesentery when it gradually sinks down after birth and comes to lie in the true pelvis.

Prostate. Above and below the opening of the primary excretory ducts, several independent glands grow out into the surrounding denser mesoderm of the wall. In the male the mesoderm develops smooth muscle fibers and dense connective tissue which mark off the structure—prostate—from the surrounding tissue, and enclose the developing glands. In the female the development of the mesoderm into smooth muscle fibers and compact connective tissue does not take place, and the prostatic tubules remain isolated structures. Few glands are formed in the female; later they may undergo development and form the paraurethral glands.

Prostatic glands begin as solid buds around the periphery of the sinus, most numerous on the dorsal surface, less so on the lateral, and rare ventrally. Simultaneous with their appearance those on the dorsal and lateral surface branch, while those on ventral surface remain unbranched and for the most part degenerate. A bilateral symmetry is apparent. Direction is cranial, only lowermost of lateral grow caudally. Two thirds of glands arise caudal to the openings of the primary excretory ducts. The prostatic glands grow into the muscle and force the muscle fibers apart, and so produce the trabecular arrangement of the musculature.

All the prostatic glands are not united in the prostate; accessory glands appear in the interval between the prostate and Cowper's glands.

Bulbo—Urethral and the Vestibular Glands (Cowper, Bartholin). Solid epithelial buds arise from the dorsal wall of the pars pelvina of the urogenital sinus. They are usually paired, two on each side; frequently one on one side and two on the other. The buds of each side grow upward from the sinus and lie at first in the compact mesoderm which forms the corpus cavernosum urethrae. The glands grow through this mantle and enlarge in the looser mesoderm between the rectum and sinus.

In the female, the glands originate as in the male with increased development later. Up to and after birth there is a slow increase of the terminal vesicles, and after puberty another rapid growth. At the climacterium Bartholin glands undergo degeneration and may be wanting in old age.

Small Sinus Glands. Glands are entodermal in origin; they appear in succession around the entire periphery of the urogenital sinus. They possess the same character as Cowper's glands except that they do not attain to the same extensive development.

PART IV

CLOACAL MEMBRANE AND ITS MESODERMAL BORDERS—BAND ON BODY STALK, EXTERNAL GENITALIA, INFRA-UMBILICAL ABDOMINAL WALL—PERINEAL MUSCULATURE, EXTERNAL CLOACA, ANAL ORIFICE, UROGENITAL ORIFICE

Cloacal Membrane and Its Mesodermal Borders. The inferior wings of mesoderm by their medial borders form the mesoderm at the lateral margins of the cloacal membrane. The remainder of the wings form the lateral walls of the internal cloaca. The mesoderm in the region of the cloacal membrane on the under surface of the umbilical cord invade this portion of the membrane from each side and unite to form a band on the under surface of the cord. The mesodermal borders of the reduced cloacal membrane become thickened and raised above the surface to form a trough with the cloacal membrane as its roof.

The band on the cord by its cranial slope forms the beginning of the infra-umbilical abdominal wall. The cephalad advance of the cord and the medial advance of the lateral abdominal wall complete the wall. The caudal slope of the band develops a projection—the genital tubercle—which carries out with it a portion of the urogenital sinus—phallic sinus. The genital tubercle is part of the band on the cord and develops in the wall derived from the band. This accounts for the superficial perineal pouch of the adult.

The genital tubercle at each side slopes into the borders of the trough which lies for a short distance on the under surface of the tubercle. The trough is the genital fossa and its borders are the genital folds. The mesoderm surrounding the reduced cloacal membrane and tubercle forms the sphincter cloacae.

The cloacal borders behind the cloacal membrane develop a tubercle—anal tubercle—at each side, which are joined by a transverse bar across the midline.

The band on the cord at the beginning of its cranial slope develops the pubic bone which forms the anterior pelvic brim and divides the perineum from the anterior abdominal wall. The band develops the musculature of the anterior wall of the bladder. This wall develops extraperitoneally and remains so throughout life.

The anal tubercle at each side advances along the lateral mesodermal border of the cloacal membrane and bends medially to meet its fellow of the opposite side. The anal tubercles fuse on the cloacal membrane at a point corresponding to the dorsal surface of the pelvic bar. The fusion of the tubercles takes place a short distance in front of the cloacal membrane. The membrane is divided by the fused tubercles into a ventral urogenital portion and a dorsal anal portion. The genital fossa is similarly divided into the urogenital portion in front and the anal pit behind. The fused tubercles form the perineal or external bar which lies on the back of the pelvic bar. The prerectal space is separated from the external surface of the perineum by the perineal bar and its contiguous pelvic bar. The sphincter cloacae, by the fusion of the tubercles, forms the urogenital sphincter and the external anal sphincter around the corresponding parts of the genital fossa. The urogenital sphincter develops in conjunction with the genital tubercle, and the anal sphincter in connection with the longitudinal muscle of the rectum.

The longitudinal muscle of the rectum grows beyond the circular muscular coat and penetrates the anal sphincter to gain attachment on the deep surface of the dermal layer. The sphincter cloacae by its anal portion is partly brought into the gut wall and by its urogenital portion into the inferior portion of the infra-umbilical wall. The two portions of the sphincter cloacae remain attached to each other throughout life. The sphincter cloacae is composed of deep and superficial portions.

The levator musculature develops with the pelvic bones, the tail, and the rectum. Secondary attachments are formed with contiguous structures; urethra, prostate, vagina. A portion of the levator musculature attached to the rectum separates on its perineal surface

and remains attached to the levator and free from the rectal wall. This is the puborectalis muscle.

The downgrowth of the longitudinal muscle of the rectum receives fibers from the levator musculature and together they penetrate the musculature of the anal sphincter. The outer margin of the penetrating muscle lies at the border between the superficial and deep portions of the anal sphincter. The penetrating muscle composed of smooth and striated fibers invades the sphincter in a fan formation. Medially the fan penetrates the inferior portion of the internal circular coat of the gut. Thickening of the internal circular coat to form the internal sphincter does not occur until the penetrating fibers have acted on the circular bundles to cause them to override each other. The internal sphincter is formed by the action of its penetrating longitudinal musculature.

The portion of the external sphincter that lies free on the penetrating fan is the deep anal sphincter or from its relation to the longitudinal musculature the extrinsic sphincter externus ani. The portion of the sphincter within the penetrating fan is the superficial sphincter or, from its relation to the longitudinal musculature, the intrinsic external sphincter ani.

The smooth internal sphincter is formed by the longitudinal penetrating muscle and is intrinsic to the longitudinal wall.

External or Ectodermal Cloaca. The urogenital cloaca before its completion receives material from its functioning kidney and the intestinal tract. (The buccopharyngeal membrane has ruptured at the end of the third week.) The corresponding portion of the cloacal membrane yields to the pressure of its accumulating contents and forms small openings during the eighth week for its passage. The separation of the ventral from the dorsal cloacae is completed by the formation of the pelvic bar. Desquamation of the entodermal lining of the cloacal membrane leaves the thin sheet of ectoderm of the cloacal membrane with its multiple openings in the urogenital area which again drain the intestinal and urinary tracts. The passage —external cloaca—extends from the upper margin of the desquamated entodermal layer of the cloacal membrane through the anterior genital fossa to the outer margin of the genital folds. The urogenital cloacal membrane disappears and the urogenital sinus is open at the surface. A thick layer of cells derived from the edge of the anal cloacal membrane persists and forms a lining on the walls of the dorsal portion of the external cloaca. The pelvic bar by its dorsal surface forms the anterior wall of the forming anal canal.

Subsequently the perineal bar forms on the back of the pelvic bar. The anal canal is separated from the anterior genital tract. The thin ectodermal sheet separating the canal from the surface ruptures during the third month, under pressure of the intestinal contents above, and the anal canal opens to the exterior. The buccopharyngeal membrane has been open for a considerable time.

The derivation of the lining of the anal canal from the ectoderm to a small extent and the entoderm to a greater extent accounts for the transitional nature of the epithelial lining of the adult anal canal.

Development of Structures of the Pelvic Outlet. The mesoderm at the borders of the cloacal membrane proliferates under cover of the ectoderm and forms an elevated margin that surrounds the genital fossa whose roof is the submerged cloacal membrane. A broad conical eminence develops anteriorly, the genital tubercle, barely separated cranially from the umbilical cord. Its rounded free end as the primordium of the glans is faintly separated from the basal portion. Lateral slopes below the glans area extend into the outlying basal tissue as the lateral buttresses which will form the future corpora cavernosa. Caudally the slope extends to a horizontal bar on either side of the cloacal membrane connecting the anal tubercles behind the cloacal membrane. The anal tubercles grow forward and converge across the fossa to fuse in the midline on the posterior surface of the pelvic bar. The genital fossa is divided by the fused anal tubercles into a posterior anal pit and an anterior urogenital sinus.

The mesoderm at the margins of the urogenital sinus develops its structures in the mesoderm that helps form the infra-umbilical abdominal wall and becomes included in that portion of the abdominal wall.

Development of the Male Genitalia. The tubercle becomes elongated and forms lateral buttresses which migrate medially to form a somewhat cylindrical phallus whose base is separated from the surrounding body areas by the newly formed labioscrotal swellings; the latter appear first as broad elevated areas extending laterally from the base of the phallus, but are soon changed into swollen ridges separated from the phallus by the lateral phallic grooves. With the elongation of the tubercle to form the phallus, the urogenital opening becomes sharply outlined and the sex distinction becomes emphasized by the length of the opening. The genital folds are now the urethral folds. In the male the opening extends from the base to the apex of the phallus, whereas in the female it extends only

to the base of the glans. The lips of the urethral folds meet in the midline to form a raphé and transform the open urogenital sinus into the tubular urethra, open at its distal portion. The labioscrotal swellings take their final position caudal to the penis to form the scrotum, the halves of which becomes approximated to form the raphé in the midventral line and maintain their bilateral character. The glans develops the wide coronary sulcus and becomes sharply defined. The gradual closure of the urethral opening develops synchronously with the growth of the prepuce. The urethral opening shifts to a more terminal position in the frenular notch of the glans during the later stages of prepuce formation. The urethral opening becomes entirely closed and later there is formed the permanent terminal urethral opening at the apex of the glans.

The prepuce is first recognized as a pair of swellings on the inside of the urethral opening. These swellings gradually fuse together over the dorsum of the shaft to form a flat skin whose distal margin envelops the corona of the glans. This fold of skin grows distally until it covers the glans completely.

Development of the Female External Genitalia. The entire process of development of the external genitalia in the female is accompanied by fewer structural changes than occur in the male. Completion of development, however, takes a longer time so that final differentiation in the female does not synchronize with the more differentiated transformation in the male. The genital tubercle of the female resembles that of the male. The genital groove, however, in the female is shorter. The groove on the tubercle becomes obliterated and the genital folds separate from the groove. Characteristic of the female is the caudal decurvation which appears to be brought about by an excess in the growth of the cavernous over the urethral regions of the phallus. The urethral folds develop as caudal projections supporting the slightly overhanging glans, which becomes more clearly defined than in the male. The caudal ends of the labioscrotal swellings grow towards each other and join in the midventral line to form the posterior commissure. In this manner these originally paired swellings are transformed into a cranially opened horseshoe-shaped rim, enclosing the rest of the external genitalia and separating them from the anus. In the female the labioscrotal swellings form the labia majora. The shaft and glans of the phallus form the clitoris, and the ununited urethral folds separate from the clitoris and become the labia minora. The unobliterated urogenital orifice becomes the urethrovaginal orifice. The prepuce is formed as in the male but its growth is slower.

PART V

INTESTINAL CANAL—INTESTINAL LOOPS—ROTATION OF COLON—
CECUM AND APPENDIX ATTACHMENTS OF MESOCOLON

Intestinal Canal. The diaphragm develops from the mesoderm of the head fold. The lateral portions developed from the closed pleuroperitoneal channels grow towards the median plane to fuse with the dorsal portions growing ventrally from the dorsal mesentery. On completion of the diaphragm, the abdominal portion of the cœlomic cavity is separated from the thorax. Incomplete fusion of the diaphragm usually on the left side leaves an opening through which a portion of the abdominal contents may pass into the thorax to form a congenital diaphragmatic hernia.

The peritoneal cavity is small. The dorsal wall of the embryo is strongly anteflexed, the space between the intestines and side walls is shallow and the greater part of its ventral aspect is taken up by the body stalk and vitelline duct. The peritoneal cavity is open to the extra-embryonic cavity.

The midgut forms a forward bend—enterocolic or first primary loop—which is attached at its apex to the vitelline duct. The primary loop is supplied by the superior mesenteric artery. The descending cranial limb of the loop is continuous with the terminal foregut whose developing stomach with its attached liver develop in the upper portion of the peritoneal cavity. The ascending caudal limb is continuous with the hindgut which terminates by expanding into the retroperitoneal entodermal cloaca. The two limbs lie side by side. The caudal limb develops a small expansion—cecum—which marks the beginning of the colon. The lengthened mesentery diminishing toward the mesogastrium above and terminating at the cloaca blow, is fan-shaped. The mesentery is attached along the middle of the dorsal wall between two elongated and rounded swellings— Wolffian bodies—which fill the dorsal wall between the mesentery and the lateral parietes.

The liver grows enormously and almost fills the peritoneal cavity. The growing intestinal tube is crowded into the extra-embryonic space between the vitelline duct and the median folds of the amniotic sac. The folds and ducts fuse by their outer coats of morula mesoderm beyond the projecting enterocolic loop and enclose a space— peritoneal space of umbilical cord—in which the ileal coils of the primary loop develop.
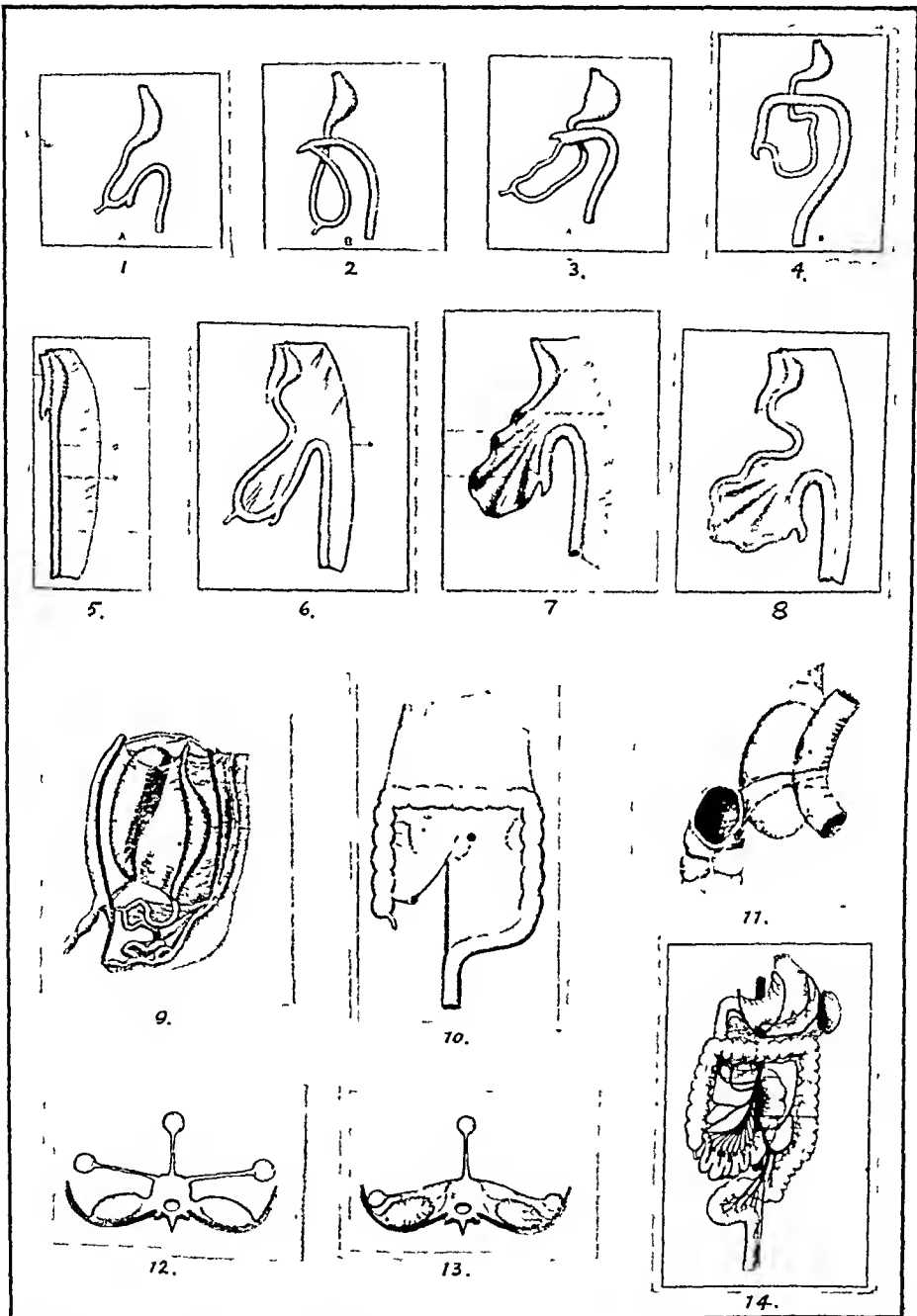
PLATE XVI. Rotation of intestinal canal. 1–4, rotation of intestinal tube. 5–8, early growth of intestinal loops and mesentery. 9, ventral and dorsal mesentery of gastrointestinal tract. 10, completed rotation of colon forming the infracolic compartment. Omentum turned up. Relation to kidneys, duodenum and superior mesenteric artery. Attachments of mesocolon shown by dotted lines. 11, right mesocolon in relation to right kidney and duodenum. 12, cross section of kidney and intestinal mesentery before fixation of lateral colon. 13, after fixation. 14, final arrangement of arteries of alimentary canal after rotation. (After Huntington.)

Second and Third Loops of the Three Primary Loops.   The cranial limb of the enterocolic loop at its origin from the tapered swelling of the stomach bends laterally, with its convexity to the right abdominal wall and forms the gastroduodenal loop supplied chiefly by a branch of the celiac artery. The caudal limb of the enterocolic loop at its termination in the hindgut, bends laterally with its convexity toward the left abdominal wall and forms the left colic loop on the posterior abdominal wall, chiefly supplied by the inferior mesenteric artery.

The enterocolic loop in the peritoneal cavity of the cord with the intra-abdominal cephalic right gastroduodenal loop and caudal left colic loops are the fundamental loops which form the mesenteric portion of the intestinal canal. The enterocolic loop undergoes an elaborate development to form the distal part of the duodenum, the jejunum, ileum, cecum, ascending colon, and the right half of the transverse colon. The base of the enterocolic mesentery receives the superior mesenteric artery from the aorta at its center where the mesentery early becomes fixed to the ventral surface of the first lumbar vertebra. The left colic loop develops later than the first two fundamental loops to form the left half of the transverse colon, the splenic flexure, the descending colon, and sigmoid colon.

A portion of the mesocolon of the left colic loop under pressure of the neighboring intestinal coils early fuses with the dorsal peritoneal wall in the region of the left side of the left kidney and there fixes its attached gut.

The attachments of the mesentery to the region of the first lumbar vertebra by its enterocolic portion and to the left iliac fossa by its left colic portion are the most constant of the fixed points formed by the mesentery.

The rapid growth of the liver subsides, the ventroflexion of the embryo becomes a dorsoflexion at the lumbosacral angle, and the space of the left abdominal cavity enlarges. The vitelline duct, compressed by the median folds of the greatly distended amniotic sac, disintegrates and loses its attachment to the intestinal canal. The midgut, free in the extra-embryonic peritoneal cavity of the cord withdraws its coils into the roomier embryonic abdominal cavity where they complete their development.

The colic portion of the primary loop withdraws last from the extra-embryonic peritoneal cavity. The colic portion ascends ventrad to the surface of the intestinal coils and crosses the duodenum to bring the cecum into the right hypochondriac region below the pyloric end of the stomach and liver. The proximal portion

of the colon lengthens to form the ascending colon and bends toward the right iliac fossa where the cecum descends ventral to the kidney, and takes its adult position during the later prenatal or early postnatal period. The cecum may fail to descend and remain high in the abdominal cavity.

The rotation of the caudal ascending limb of the primary loop from a position below and to the left of the upper limb to a position above and to the right of the upper limb twists the mesentery of the loop around its axis of the superior mesenteric artery. A further rotation of the proximal ileocolic portion carries the ileal coils over the cecum and the ileum enters the cecum from the left side.

The colic portion of the twisted mesentery fuses with the posterior abdominal wall of the right side from the superior mesenteric artery on the duodenum to the cecum. Left of the artery the mesentery fuses with the ventral surface of the duodenum from which the line of fusion extends laterally to the anchorage of the descending colon. The voluminous intestinal coils crowd the ascending and descending colon laterally against the right and left lumbar walls. The apposed serous surfaces of the lateral colon, mesocolon and peritoneal wall fuse and the attached portions of the gut are retroperitoneal in the area of fusion. The retroperitoneal portion of the gut on its lateral surface is covered by an avascular sheet of peritoneum. The fused mesocolon helps form the prerenal peritoneum at each side.

The colon between its fixed regions in the left and right iliac fossae, stretches across the abdominal cavity as the transverse colon. The great omentum descends in front of the transverse colon and the apposed serous surface of the deep layer of the omentum fuses with the cephalic portion of the colon and mesocolon.

The colon in the left iliac fossa below its attached descending portion, bends to form a small loop—sigmoid loop—whose upper limb begins at the lower end of the descending colon and whose lower limb ends at the colon derived from the cloaca.

The sigmoid mesocolon partly retains its primitive vertical origin to the dorsal midline and partly extends horizontally to the fixed descending mesocolon. The vertical and horizontal lines of attachment meet at a right angle open inferiorly and to the left, to form the intersigmoid fossa. Deep to this angle of mesenterial attachment lies the left ureter.

The cecum first appears as a circumscribed thickening of the lower limb of the enterocolic loop. The thickening forms a conical pouch which becomes narrow at its apex where a narrow tubular extension of the appendix develops. The pouch of the cecum grows

beyond the apex and the appendix becomes a diverticular appendage. The three longitudinal muscular bands meet at the root of the appendix.

The colon, from the lateral fixation of its ascending and descending portion continues medially with the fixation of the cranial leaf of the transverse mesocolon to the omentum, forms the infracolic compartment of the posterior wall of the peritoneal cavity. The mesentery of the small intestines lies within the compartment.

PART VI

WALL OF COLON, RECTUM, AND ANAL CANAL—EPITHELIAL LINING, MUSCULAR WALL, GLANDS

Wall of Colon, Rectum, and Anal Canal. The colon or large intestine begins with the cecal swelling on the caudal limb of the enterocolic loop and ends with its attached dorsal mesentery at the expansion of the hindgut that forms the internal cloaca. The rectum, upon the division of the internal cloaca, begins at the termination of the mesenteric gut and ends on the entodermal or internal surface of the cloacal membrane. The anal canal begins at the entodermal surface of the cloacal membrane and ends at the inferior margin of the anal pit on the surface of the perineum to communicate with the exterior.

The changes that take place in the entodermal epithelial tube—mucous membrane—of the appendix, colon, and upper rectum are similar to those of the esophagus, stomach, and small intestines. The wall of the mesenteric colon, from within out, consists of the stratified epithelium surrounded by a zone of loose mesoderm bounded by a thin layer of forming muscle cells, the circular muscle wall; the circular layer is covered by a layer of mesoderm and the entodermomesodermal tube is invested by mesothelium excepting at the area of attachment of the mesentery.

Beginning in the upper rectum, the epithelium becomes thickened to form longitudinal ridges. The mesodermal wall indents the lumen and the ridges become folds of the entire wall. The process extends to the descending colon, and before extension to the left transverse colon occurs, a similar process begins at the ascending colon near the ileocolic valve. The transverse colon develops its folds last. The epithelium outside the folds develop conical projections which are the transitory villi of the colon. The sigmoid colon at the crest of its ridges develops goblet cells which are more numerous than in the descending colon later. At various points the folds fuse at their tops
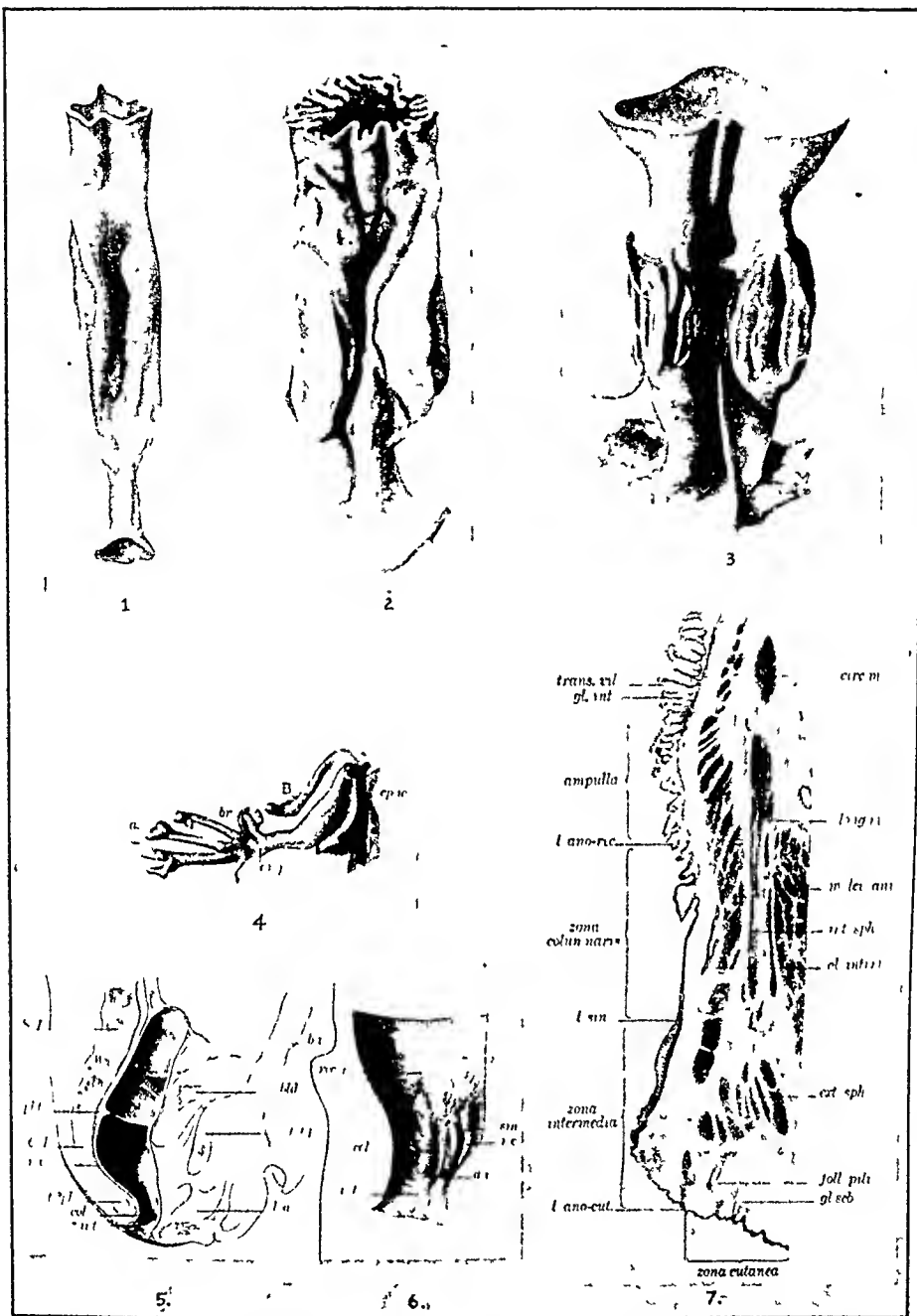
PLATE XVII. 1, 2, 3, reconstruction of epithelium of the pars analis recti of an embryo of 240 mm. Note in 3 slender extension from each side of epithelial tube is an intramuscular gland. 4, branched intramuscular gland in embryo of 245 mm. *a*, acinous termination. *br.*, branch. *amp.*, ampulla. *ep.w.*, epithelial wall. 5, sagittal section through pelvis at birth. *S.I.*, first sacral vertebra. *pl.tr.*, plica transversalis recti. *C.I.*, first coccygeal vertebra. *rec.a.*, rectal ampulla. *ext.spb.*, external sphincter muscle. *z.col.*, zona columnaris. *z.int.*, zona intermedia. *b.w.*, body wall. *bld.*, bladder. *sym.p.*, symphysis pubis. *l.a.*, levator ani muscle. 6, sagittal section through the lower part of the rectum. *rec.a.*, rectal ampulla. *z.col.*, zona columnaris. *z.int.*, zona intermedia. *sin.*, rectal sinus. *r.c.*, rectal column. *a.v.*, anal valve. 7, longitudinal section through the pars analis recti. *trans.vil.*, transitory villus of the ampulla. *gl.int.*, intestinal gland. *ampulla*, ampulla recti. *l.ano-rec.*, anorectal line. *l.sin.*, sinuous line. *l.ano-cut.*, anocutaneous line. *circ.m.*, and *long.m.*, circular and longitudinal layers of the muscularis. *m.lev.ani*, levator ani muscle. *int. spb.*, internal sphincter muscle of the anus. *ext.spb.*, external sphincter muscle of the anus. *gl.intrm.*, intramuscular gland. *foll.pili*, hair follicle. *gl.seb.*, sebaceous gland. (After Johnson.)

and form epithelial cysts. The epithelium degenerates and the cysts disappear particularly in the appendix and cecum. In the rectum, the cysts open to the lumen and become glands again.

The epithelium develops knobs of growth into the mesoderm which become the intestinal glands. The glands multiply by splitting longitudinally.

Under the distending pressure of the accumulating meconium the villi become flattened out and disappear; the mucosa becomes thinned out and the glands are spread apart and shortened.

Lower Rectum and Anal Canal: Anorectal Canal. The lower rectum, where it passes into the anal canal, develops ridges and folds which are transverse in direction. The division between the rectum and anal canal corresponds to the level of the pelvic floor. Early the rectal tube develops a spindle-shaped swelling which is elongated at first and then becomes more transverse at the bulbus analis and is the site of the rectal columns. Later, below the bulbus a second swelling appears—bulbus terminalis—limited inferiorly by the anal membrane, and is the site of the future zona intermedia.

The bulbus analis develops three longitudinal folds which extend throughout its length and end below on the zona intermedia. The posterior and lateral portions are each folded. Secondary folds develop in considerable numbers and obscure the primary folds. The larger infoldings of the mucosa form the rectal columns—columns of Morgagni—and are present in great numbers. Sixty were counted by Johnson in a 240 mm. embryo. The smaller folds are similar in structure to the more conspicuous columns. The columns, at their inferior portion, are joined together by a tag—anal valve—and the depression between the raised columns—rectal sinus—continues below their union to form the recess of the crypt.

Early the pars analis is lined by a few layers of low columnar epithelium similar to the epithelium of the early digestive tract. This zone of epithelium marks the transition of the mucosa of the intestinal tract above to the skin of the surface below. This transition passes through two superimposed areas to reach the skin margin— anocutaneous border—the zona columnaris, and the zona intermedia. The simple columnar epithelium becomes the stratified columnar epithelium of the zona columnaris. The stratification consists of two to three layers, and extends above for a short distance on the rectum to form the anorectal line. Below, the stratification extends at various points into the anal skin folds which extend inward and upwards from below.

The zona columnaris, at its rectal sinuses, develops tubular glands which lie in the submucosa. Six or eight of the simple tubular

glands become branched and penetrate the neighboring muscular wall to form the intramuscular glands. The main ducts of the gland extend outward and usually downwards, and penetrate the internal sphincter muscle where a swelling of the duct gives off several tubular branches that end blindly in the intramuscular connective tissue. Occasionally a tubule penetrates the outer longitudinal muscle. An occasional tubule forms acini. A small amount of lymphoid tissue forms around the terminations of the tubules. Stratified cuboidal epithelium is found in the larger ducts of the intramuscular glands, and simple cuboidal epithelium in the branches of the duct.

The intramuscular glands show evidence of secretory activity in the fetus and at birth.

The zona intermedia is lined by stratified squamous epithelium composed of several layers of the polygonal cells forming a thicker layer than the cells of the epidermis. Dermal papillae are present but hairs and sweat glands are absent. In the lower part of this zone are occasional sebaceous glands without hairs, and the epithelium is slightly cornified. The transition of the epithelium from the zona columnaris above is abrupt. The change in epithelium occurs at the level of the anal valves from which the changing level extends upwards on the summits of the rectal columns. The abrupt transition takes place along a zigzag line—anocutaneous line of Herrmann, or linea sinuosa analis. The zona intermedia, below, gradually goes over into the skin, and forms with the skin the anocutaneous line which is marked in the skin by the first hair follicles. The anocutaneous line is not well marked. The skin of the immediate neighborhood of the anus is the zona cutanea and is characterized by large sweat glands—the circumanal glands of Gay.

The muscularis mucosae of the rectum is similar to the colon above. Early, the muscularis mucosae extends into the zona columnaris as a thin sheet of fibers lying close to the epithelium. Later, fibers of striated and smooth muscle appear in the submucosa. The striated fibers of the rectal wall are terminal fibers of the rectal levator muscle.

The submucosa becomes fibrous. Numerous nerve fibers, blood vessels and Paccinian corpuscles are present in the submucosa.

The internal circular and outer longitudinal muscular coats of the colon continue downward to form complete muscular coats of the rectum. The inner circular muscle ends abruptly below. The outer longitudinal muscle, continuous around the gut, becomes thickened into bands at the antimesenteric and lateral borders to form the teniae coli. The teniae extend from the base of the appendix to the

end of the mesentery at the beginning of rectum. The outer longitudinal coat completely invests the rectum and by its external surface is attached in front to the pelvic bar, laterally and posteriorly to the levator musculature, and posteriorly at its in inferior termination to the rectococcygeus muscle. Above the longitudinal coat of the rectum is covered on its anterior and lateral surfaces by peritoneum, middle portion only anteriorly, lower portion extraperitoneally, the posterior surface is entirely extraperitoneal. The posterior surface of the rectal tube separates from the anterior surface of the sacrum when the longitudinal muscle is formed there. The separation of the gut from the sacrum leaves a space—retrorectal space—between them. The retrorectal space is bounded inferiorly by the attachment of the longitudinal muscle to the rectococcygeus muscle from the lowest piece of the sacrum. Above, the space is bounded by the lower branches of the inferior mesenteric artery. The outer longitudinal muscle of the rectum with fibers from its attached levator muscle continues beyond the internal circular coat below and spreads out in a fan. The muscular fibers of the fan end in small connective tissue fibers which penetrate the smooth internal circular muscle and the external sphincter muscle; and as septae of the penetrated musculature divide them into bundles. The septae attach to the lining of the anal canal. The internal circular and the external sphincter bundles are attached to the angles of the fan and enclosed within the space formed by the limbs of the longitudinal muscle and the covering deep surface of the anal lining. The internal circular muscle attached to the penetrating longitudinal muscle forms the internal or smooth sphincter. The internal circular fibers extend to the constriction between the bulbus analis and bulbus terminalis, and later when the bulbus terminalis disappears, extends into the region of the zona intermedia. The external sphincter surrounds the middle part of the zona intermedia.

The rectum develops infoldings of its mucous membrane to form transverse rectal folds. The folds become more pronounced and with further growth of the rectum become spread apart.

Early, all of the pars analis recti lies between the third sacral vertebra and the tip of the coccyx. At birth the pars analis lies below the tip of the coccyx.

Upon the completion of the development of the anal canal, external genitalia, and infra-umbilical abdominal wall from the mesodermal borders of the cloacal membrane and the band on the under surface of the umbilical cord, the developed structures preserve evidence of their common origin in the form of attachments to each

other. Similarly, the division of the internal cloaca into rectal and urogenital portions with the formation of the pelvic cavity preserves evidence of their common origin. The pelvic cavity is in relation to the attachments formed from the internal cloaca and the external cloaca. The urogenital and the rectal divisions remain joined by the pelvic bar at the inferior boundary of the pelvic cavity. The pelvic bar is contiguous by its external surface with the region of the fused anal tubercles which forms the continuation of the anal musculature into the urogenital musculature. The urogenital musculature with its attached urogenital organs develops in association with the lowermost portion of the infra-umbilical abdominal wall. The wall with the deep portion of the urogenital sphincter cloacae, forms a space within which are confined the structures of the urogenital sinus. The space is the superficial perineal pouch.

Muscle is a differentiated product of mesoderm whose less differentiated portion forms the connective tissue envelope for the muscle fiber. Degeneration of muscle forms aponeurosis, fascia, and tendon. The final form of a muscle fiber, striated or unstriated, is the resultant of tensional stresses.

More Common Malformations of the Anorectal Canal.   Usually the intestinal tract opens on the inferior surface of the embryo by a continuous canal. Infrequently, the lower passage is discontinuous or communicates with the nearby urogenital passage.

Failure of the pelvic bar to fuse with the cloacal membrane leaves a fistulous communication between the rectum and bladder. Invasion of the entodermal wall of the rectum by mesoderm obliterates the lumen and reduces the entodermal tube to a ligament.

Incomplete union of the anal tubercles leaving a shallow groove between them, followed by union over the groove by the labioscrotal folds, results in a fistulous opening in the anal canal that communicates with the vestibule of the vagina or penile urethra.

The cloacal membrane invaded by mesoderm forms a persistent fibrous membrane blocking the outlet of the anal canal.

REFERENCES

General References

AREY, L. B. Developmental Anatomy. Philadelphia, 1924. Saunders.

BROMAN, I. Die Entwicklung des Menschen vor der Geburt, pp. 45, 199, 201. Munich, 1927.

BROMAN, I. Normale und abnormale Entwicklung des Menschen. Wiesbaden, 1911.

BRYCE, T. A., and TEACHER, J. H. Contributions to the Study of the Early Development and Imbedding of the Human Ovum. Glasgow, 1908.

BEER, G. R. de. An Introduction to Experimental Embryology. Vertebrate Zoology. Oxford, 1928. Oxford Clarendon Press.

FRAZER, J. E. Manual of Embryology. London, 1931. Baillière, Tyndall & Cox.

HAECKEL, ERNST. The Evolution of Man, vol. 1. Translated by Joseph McCabe. New York, 1905. G. P. Putnam's Sons.

HERTWIG, OSCAR. Elemente der Entwicklungslehre, 4th ed. Jena, 1910.

HIS, WILHELM. Anatomie menschlicher Embryonen. Atlas 1–111. 1860.

HUNTINGTON, A. S. Human Peritoneum and Abdominal Cavity. New York, 1903.

JORDAN, H. E., and KINDRED, J. E. A Textbook of Embryology, 3rd ed. New York, 1937. D. Appleton-Century.

KEIBEL and MALL. Human Embryology. Philadelphia, 1912.

KEITH, ARTHUR. Human Embryology and Morphology, 4th ed. London, 1921.

KOLLMAN, J. Hand Atlas der Entwicklungsgeschichte des Menschen. Jena, 1907.

LEWIS, W. H. Gray's Anatomy, 23rd ed. Philadelphia, 1936. Lea & Febiger.

MORGAN, T. H. Experimental Embryology. New York, 1927. Columbia University Press.

MINOT, C. S. Human Embryology. New York, 1892.

NEEDHAM, JOSEPH. A History of Embryology. London, 1934.

ROBINSON, ARTHUR. Cunningham's Textbook of Anatomy, 6th ed. Baltimore, 1936. William Wood.

Special References

BING, ROBERT. Ueber angeborenis Muskeldefekte. Basel Thesis. Berlin, 1902. G. Reiner.

BALL, C. B. The anal valves, their origin and pathogenic significance. *Matthews Med. Quart.*, vol. 1, 1894.

BÉRAUD, M. Du mode de terminaison des fibres longitudinales du rectum. *Bull. et mém. Soc. de biol.*, 4: 163–166, 1858.

BLOOMFIELD and FRASER. Development of the lower end of the vagina. *Am. J. Anat.*, 62: 9–32, 1927.

BRAUN, W. O. Untersuchungen ueber das Tegument der Analoeffnung. Inaug. Diss., Koenigsberg, pp. 1–50, 1901.

BRYCE, THATCHER. Observation on early development of the human embryo. *Tr. Roy. Soc. Edinburgh*, 1924.

CAREY, E. J. Studies in the dynamics of histogenesis. Tension of differential growth as a stimulus to myogenesis. *Am. J. Anat.*, 29: 93–115; 341–377, 1921.

FELIX, W. The development of the urinogenital organs. In Keibel and Mall. Human Embryology, 2: 787, 921, 960. Philadelphia, 1912.

FETZER, M., and FLORIAN, J. Cloaeal membrane in earliest embryos. The youngest human embryo (Embryo Fetzer) with a cloacal membrane. *Anat. Anz.*, 67: 381–492, 1929.

FLORIAN, J., and VÖLKER. Development of the primitive streak, the cloaeal membrane, and the allantois in the human. *Ztscbr. f. mikr. Anat.*, 16: 75–100, 1928.

CHIARI, H. Ueber die analen Divertikel der Rectumschleimhaut und ihre Beziehung zu den Analfisteln. *Med. Jabrb.*, pp. 419–427, 1878.

GAY, A. Die Circumanaldrusen des Menschen. *Sitz.-ber. d. Kais. Akad. d. Wiss.*, 63: 329–331, 1871.

HART, D. B. A contribution to the morphology of the human urinogenital tract. *Brit. M. J.*, 2: 773–776, 1901.

HART, D. B. Prepuce. *J. Anat. & Physiol.*, 42: 50, 1908.

HART, D. B. Hymen. *Edinburgh M. J.*, 6: 577, 1911.

HART, D. B. Development of vagina. *Brit. M. J.*, Dec. 17, 1904.

HEBRANT, G. Sur les glandes anales du chien; anatomie, physiologie, pathologie. *Ann. méd. vét.*, 48: 633–641, 1899.

HERRMAN, G. Structure and development of anal musculature. *J. Anat. et physiol.*, 16: 434–472, 1880.

HERRMANN, G., and DESFOSSES, L. On the muscle of the cloacal region of the rectum. *Compt. rend. Acad. de sc.*, 90: 1301, 1880.

HERZOG, F. Beiträge zur Entwicklungsgeschichte und Histologie der männlichen Harnröhre. *Arch. f. mikr. Anat.*, 63: 710–747, 1904.

HEUSER, C. H. A presomite human embryo with a definite chorda canal. *Contrib. Embryol.*, 23: 251–267, 1932.

HILL and FLORIAN. Head process; prochordal plate; unrelated to cloacal membrane. *J. Anat.*, 65: 242, 1929.

HUNTER, R. H. Observations on the development of the human female genital tract. *Contrib. Embryol.*, 22: 91–108, 1930.

INGALLS, N. W. A human embryo before the appearance of the myotomes. *Contrib. Embryol.*, 7: 111–134; 7–81, 1918.

JOHNSON, F. P. The development of the mucous membrane of the oesophagus, stomach, and small intestine in the human embryo. *Am. J. Anat.*, 10: 521–561, 1910.

JOHNSON, F. P. The development of the mucous membrane of the large intestine and the vermiform process. *Am. J. Anat.*, 14: 187–233, 1913.

JOHNSON, F. P. Effects of distention of the intestine upon the shape of the villi and glands. *Am. J. Anat.*, 14: 235–250, 1913.

JOHNSON, F. P. The development of the rectum in the human embryo. *Am. J. Anat.*, 16: 1–57, 1914.

JOHNSON, F. P. A human embryo of 24 pairs somites. *Contrib. Embryol.*, 6: 19, 1917.

JONES, W. F. Nature of the malformations of the rectum and urogenital passages. *Brit. M. J.*, 2: 1630–1634, 1904.

JONES, W. F. Delimitations of the rectum and its subdivisions. *Proc. Roy. Soc. Med.*, vol. 4, 1911.

JONES, W. F. Development of the vagina. *J. Anat.*, 48: 268, 1914.

JONES, W. F. Prepuce. *Brit. M. J.*, Jan. 15, 1910.

JONES, W. F. Explanation of a recto-urethral anomaly and some points in normal anatomy. *Lancet*, 2: 860, 1915.

KEITH, A. Malformation of human body from new point of view. *Brit. M. J.*, 1: 489–491, 1932.

KOFF, A. K. Development of the vagina in the human fetus. *Contrib. Embryol.*, 24: 59–91, 1933.

KUNITOMO, K. The development and reduction of the tail and of the caudal end of the spinal cord. *Contrib. Embryol.*, 7–8: 163–196, 1918.

LEWIS, F. T. The early development of the entodermal tract and the formation of its subdivisions. In Keibel and Mall. Manual of Human Embryology, vol. 2. Philadelphia, 1912.

MILJSBERG, W. A. Ueber die Entwicklung der Vagina des Hymen und des Sinus urogenitalis beim Menschen. *Ztschr. f. Anat. u. Entwick.*, 74: 684–758, 1924.

MLADENOWITSCH, L. Vergleichende anatomische und histologische Untersuchungen ueber die Regio analis and das Rectum der Haussäugetiere. Inaug. Diss., Leipzig, 1907.

NAGEL, R. Ueber die Entwicklung des Uterus und der Vagina beim Menschen. *Arch. f. Mikr. Anat.*, 36: 620–622, 1891.

NAGEL, W. Ueber die Entwicklung der Urethra und des Dammes beim Menschen. *Arch. f. mikr. Anat.*, 40: 264–287, 1892.

NAGEL, W. Ueber die Entwicklung der inneren and äusseren Genitalien. *Arch. f. Gynäk.*, 45: 453–477, 1894.

NAGEL, W. Entwicklung und Entwicklungsfehler der weiblichen Genitalien. *Veits. Handb. d. Gynäk.*, 1st ed., 1: 519–628. Wiesbaden, 1897.

OTIS, W. J. Anatomische Untersuchungen am menschlichen Rectum. Leipzig, 1887.

OTIS, W. J. Die morphogenese und Histogenese des Analhöckers nebst Beobachtungen über die Entwicklung des Sphincter ani externus beim Menschen. *Anat. Hefte*, 30: 202–258, 1905.

PATERSON, A. M. The form of the rectum. *J. Anat. & Physiol.*, 43: 127–133, 1908.

PETERS, H. Ueber die Einbettung des menschlichen Eies. Leipzig & Vienna, 1899.

PILLIET, A. Nôte sur la présence de corpuscules de Pacini dans la muqueuse anale de l'homme. *Bull. Soc. anat. de Paris*, 67: 315, 1892.

POHLMAN, A. G. The development of the cloaca in human embryos. *Am. J. Anat.*, 12: 1–26, 1911.

POPOWSKY, J. Zur Entwicklungsgeschichte der Dammuskulatur beim Menschen. *Anat. Hefte*, 12: 13–48, 1899.

RETTERER, E. Du développement de la région anale des mammifères. *Compt. rend. Soc. de biol.*, 2: 51–54, 1890.

RETTERER, E. Du développement du prépuce, de la couronne du glans et du col du pénis chez l'embryon humain. *Compt. rend. Soc. de biol.*, 2: 528, 1890.

RETTERER, E. Du rôle de l'épithélium dans le développement des organes génito-urinaires externes. *Compt. rend. Soc. de biol.*, 57: 1, 1040, 1905.

RETTERER, E. Du développement et de la structure des raphés des organes génito-urinaires. *Compt. rend. Soc. de biol.*, 57: 2, 22–25, 1905.

RETTERER, E. Développement et histogénèse comparée des organes génitaux externes. *J. d'urol. méd. et chir.*, 6: 157–173, 1914.

ROBIN, C., and CADIAT. Sur la structure et les rapports des téguments au niveau de leur jonction dans les régions anale, vulvaire, et du col utérin. *J. Anat. et physiol.*, 10: 589–620, 1874.

ROUX, C. Beiträge zur Kenntniss der Aftermuskulatur des Menschen. *Arch. f. mikr. Anat.*, 19: 721–732, 1881.

SHIKINAMI, J. Detailed form of the Wolffian body in human embryo of first eight weeks. *Contrib. Embryol.*, 18: 46–61, 1926.

SPULER, A. Entwicklungsgeschichte des weiblichen Genitalapparatus. *Handbuch d. Gynäk.*, vol. 1, p. 470, Veit-Stoeckel.

STREETER, G. L. Factors involved in the formation of the filum terminale. *Am. J. Anat.*, 25: 1–11, 1919.

STREETER, G. L. A human embryo (Mateer) of the presomite period. *Contrib. Embryol.*, 9: 389; 424, 1920.

STREETER, G. L. Miller ovum. *Contrib. Embryol.*, 18: 31–48, 1926.

STREETER, G. L. Development of the mesoblast and notochord in pig embryos. *Contrib. Embryol.*, 19: 73–92, 1927.

TANDLER, J. Entwicklungsgeschichte und Anatomie der weiblichen Genitalien, p. 8. Wiesbaden, 1913.

TAUSSIG, F. G. The development of the hymen. *Am. J. Anat.*, 8: 89–106, 1908.

TOURNEUX and LEGAY. Sur le développement de l'utérus et du vagin envisagé principalement chez le foetus humain. *J. anat. et physiol.*, 20: 330, 1884.

TOURNEUX, F. Sur les premiers développements du cloaque, du tubercule génital et de l'anus. *J. anat. et physiol.*, 1888.

TOURNEUX, F. Sur le développement et l'évolution du tubercule génital chez le foetus humain dans les deux sexes, avec quelques remarques concernant le développement des glandes prostatiques. *J. anat. et physiol.*, 25: 229–308, 1889.

TOURNEUX, F., and HERRMANN, G. Persistence of vestigial spinal cord, its role in sacro-coccygeal congenital tumor. *J. anat. et physiol.*, 23: 498–592, 1887.

TOURNEUX, M. F. Mécanisme suivant lequel s'opèrent la disjonction du rectum d'avec le bouchon cloacal, et la formation de l'anus, chez l'embryon du mouton. *Compt. rend. Soc. de biol.*, 2: 207–211, 1890.

WALDEYER, W. Beiträge zur Kenntniss der Lage der weiblichen Beckenorgane. Bonn, 1892.

WOOD-JONES, F. Some points in the nomenclature of the external genitalia of the female. *J. Anat. & Physiol.*, 148: 73–80, 1913.

WOOD-JONES, F. The morphology of the external genitalia of the mammals. *Lancet*, 1017: 1099–1103, 1914.

Wyburn, G. M. Development of infra-umbilical portion of abdominal wall; remarks on etiology of ectopia vesicae. *J. Anat.*, 71: 201–231, 1936–1937.

Miscellaneous References

Brenneman, J. Simple congenital anorectal stricture with megacolon in early infancy. *J. A. M. A.*, 89: 662–666, 1927.

David, V. C. Congenital stricture as cause of infantile megacolon. *Surg., Gynec. & Obst.*, 37: 197–201, 1923.

Fraenkel, L. Atresia ani hymenealis *Monatschr. f. Geburtsb. u. Gynäk.*, 59: 155–157, 1922.

Florian, J. Early development of man with special reference to the development of the mesoderm and cloacal membrane. *J. Anat.*, 67: 263–267, 1933.

Helwig, F. C. Imperforate anus with exit through prostatic urethra. *Am. J. Dis. Child.*, 38: 559–561, 1929.

Keibel, F. Entwicklungsgeschichte des menschlichen Urogenitalapparatus. *Arch. f. Anat. u. Physiol.*, 55–156, 1896.

Keith, A. Three demonstrations on malformations of the hind end of the body. *Brit. M. J.*, 2: 1736–1741; 1804–1808; 1857–1861, 1908.

Reichel, P. Die Entwicklung des Darmes und ihre Bedeutung fur die Enstehung gewisser Missbildungen. *Ztschr. f. Geburtsb. u. Gynak.*, 14: 82–94, 1888.

Schimkewitsch, W. Lehrbuch der vergleichenden Anatomie der Wirbeltiere, pp. 547–565. Stuttgart, 1910.

Spaulding, M. H. The development of external human genitalia in the human embryo *Contrib. Embryol.*, 13–14: pp. 67–88, 1921.

Tench, E. M. Development of anus in the human embryo. *Am. J. Anat.*, 59: 333–343, 1936.

EDITORIAL

THE PASSING OF AN ERA

WITH the death of William Mayo, closely following the passing of his brother, the curtain has fallen on an era in American medicine. Many men great in our profession have left the scene during the past two decades. For the most part they were born shortly before or immediately after the Civil War, and in their time saw the greatest changes in medical knowledge and practice since the time of Hippocrates.

Their early years were spent in the horse and buggy school, their later ones in the era of scientific medicine. They lived through the beginnings of aseptic surgery, the introduction of anesthesia; they were in their prime when a specific was discovered for the treatment of syphilis and x-rays and radium were introduced as diagnostic and therapeutic agents. They saw radical changes in medical education and enormous enlightenment in standards of medical teaching. Fly-by-night medical schools, existing merely to make money for their owners, were put out of business, and grading and classification of medical schools made possible a new excellence in facilities available and graduates produced.

Medical organizations during this period have knit together the professional group for scientific, social and public purposes. The American College of Physicians and the American College of Surgeons, the specialty boards certifying qualified practitioners in the various branches of medicine have improved standards in their own fields. Young men are finding it possible to achieve post-graduate training in the United States, without traveling abroad for such work.

William and Charles Mayo in this period founded a great clinic. They were responsible for creating a significant departure in administration, in establishing a functioning diagnostic mechanism based on co-operation, and in developing many new, important and fundamental advances in surgery. The influence on American medicine of their venture and the changes it produced, cannot yet be estimated with any accuracy, for that influence is continuing and spreading throughout the country. Scientific medicine has saved lives; the span of life is greater; each year a new startling development is announced indicating conquest of another natural enemy. Radical and even greater changes are, however, at present awaiting us.

These are troubled times. Once medical meetings were held to hear the well informed discuss scientific subjects. Now many urge that evenings be given over entirely to discussions of medical economics and the social phases of present day

practice. Apparently to many members of the profession these are topics of greater moment than the run of medical developments. We are beset by politicians, economists, reformers, social workers and others—all of whom have programs for the renovation of the present medical set-up. To the doctors it often seems as if they could remodel better to meet the new and changing order if they were abandoned by the social planners, at least for a short time.

We are definitely facing a new era in medicine. Perhaps the organizational work of the Mayos may prove as important in that new era as their scientific and technical contributions did in the one now closing. In the attempt to make medicine meet the needs of a great people, we shall find a place for the best brains among the younger men to pioneer in developing new advances in healing and in extending those advances to all. There undoubtedly are among the youth of today the names which will rank in tomorrow's records with the great names of the old era: Billings, Ochsner, Polak, Murphy, Cragin, Osler, Welch, Coffey and the rest. The Mayos have bridged a great gap. We feel justified in saying—an era in medicine has ended with their death.

T. S. W.

TUMEFACTIONS OF THE ABDOMINAL WALL*

WILLIAM H. MEADE, M.D. AND W. ROGERS BREWSTER, M.D.

Instructor in Surgery, Tulane University School of Medicine

Assistant Professor in Clinical Surgery, Tulane University School of Medicine

NEW ORLEANS, LOUISIANA

ABDOMINAL wall tumefactions irrespective of size offer considerable difficulty in diagnosis. Their etiology, anatomic origin, histologic character, the differential diagnosis and the prognosis are of concern to the profession in general and to the surgeon in particular.

The relatively small number of abdominal wall masses reported is undoubtedly due to lack of interest or a failure to appreciate the importance of these tumefactions. Whereas MacFarlane[1] of Edinburgh reported a fibrous tumor of the abdominal wall in 1835, and Muller,[2] in 1838, described a similar one which he called a "desmoid" because of its tendinous appearance, it was not until 1864, following the report of a papillary tumor of the ovary in an abdominal wall scar, by Wagner,[3] that apparently there was an increase in the interest in lesions of the abdominal wall, for there were subsequently more reported cases.

The most extensive paper on abdominal wall neoplasms is that of Klot,[4] published in 1921. In this article are reported eight personal cases which are analyzed together with the collected cases of Pfeiffer[5] 300 and Ledderhose[6] (100), all of which were of connective tissue origin.

The incidence of abdominal wall neoplasms is greater than one would commonly suspect. Whereas Gurlts[7] found in 16,637 consecutive neoplasms of all types but thirteen carcinomas and fourteen sarcomas

of the abdominal wall, approximately 0.16 per cent, in our ward there were three in 1,460 admissions, or 0.2 per cent. These were 1 per cent of 296 tumefactions admitted over a period of three years. We are reporting forty-one cases of tumor of the abdominal parietes, collected from records of Charity Hospital and Touro Infirmary. Four of these cases were personally observed.†

Because of the lack of any workable classification, we suggest the following which has as its background the etiology of the mass, i.e., congenital, irritational, and neoplastic.

  I. Congenital
    A. Benign
      1. Ectodermal
        (a) Dermoids
        (b) Moles
      2. Endodermal
        (a) Omphalomesenteric duct tumors and cysts
        (b) Urachal tumors and cysts
    B. Malignant
      1. Carcinomatous change in the above
  II. Irritational
    A. Trauma
      1. Myositis ossificans
      2. Osteoma
      3. Carcinoma, the result of thermal burns or irradiation

† Case from Touro Infirmary not included in statistics.

4. Hematoma
5. Foreign body (Schlofferis tumor, talcum granuloma)
B. Infection
  1. Nonspecific granuloma
  2. Syphilis (gumma)
  3. Tuberculosis (secondary to abdominal)
  4. Parasitic
    (a) Animal (hydatid cyst)
    (b) Vegetable (actinomycosis)
III. Neoplastic
A. Ectodermal
  1. Benign
    (a) Papilloma
  2. Malignant
    (a) Basal cell carcinoma
    (b) Squamous cell carcinoma
    (c) Melanocarcinoma
B. Mesodermal
  1. Benign
    (a) Fibroma (simple, desmoids, keloids)
    (b) Lipoma
    (c) Myoma
    (d) Fibromyoma
    (e) Degeneration forms of the above
    (f) Angiomata
    (g) Neurofibroma
    (h) Osteoma
    (i) Chondroma
  2. Malignant
    (a) Fibrosarcoma
    (b) Sarcoma (spindle and round cell)
    (c) Melanosarcoma
    (d) Angiosarcoma
    (e) Endothelioma
    (f) Osteosarcoma
C. Endodermal
  1. Benign
    (a) Endometrioma
  2. Malignant
    (a) Metastatic carcinoma of the gastrointestinal tract
    (b) Metastatic carcinoma of the genitourinary tract

As will be seen later, many masses are a combination of these. They are either primary in the wall or secondary to pathologic lesions in the abdominal cavity or other primary sites.

In our series, there were sixteen desmoids, three neurofibromas, two spindle cell sarcomas, one round cell sarcoma, two fibrosarcomas, one myxosarcoma, one melanosarcoma, one lipoma, one osteolipoma, one hematoma followed by a fibroma, one hematoma secondarily infected, two nonspecific granulomas, three gummas, one dermoid, and four metastatic malignancies, one each from the stomach, colon, testicle, and gall-bladder. There is no doubt in our minds that there are more cases in the hospital record which we were unable to find, and therefore the comparison to the total admissions or the consecutive admissions of malignancy would be of little value.

In a survey of the literature, it is seen that the most common abdominal wall neoplasm is of mesodermal origin. (Figs. 1 and 2.) The fibromas and fibrosarcomas with their varying microscopic pictures are the predominant type (Table 1) and apparently occur at any age, but most commonly between the ages of 20 and 40. The female is affected six times as frequently as the male (Fig. 2), and the para is more susceptible than the nullipara. (Fig. 3.) Of particular interest are those tumors which occurred in four children under the age of 10. (Table 1.)

The occurrence of abdominal wall neoplasms postpuerperally is a point in favor of traumatic origin, as there is no question that the abdominal musculature is exercised to the point of rupture during labor. In our series, as in Klot's, the relationship of abdominal wall tumors to pregnancy is apparent. The question of trauma as an etiologic factor is controversial as there is no scientific evidence to substantiate traumatotropism.

Desmoids or fibromas of the rectus muscle and its sheath are of particular interest not only because of their higher postpartal

incidence (Fig. 3), and their infrequent occurrence in operative wounds[50,46] (1 in 100,000 operative wounds), but also because complete eradication is necessary to

vestigation should be made for metastases. In our series, which includes primary and metastatic masses, 22.5 per cent were malignant. (Fig. 6.)
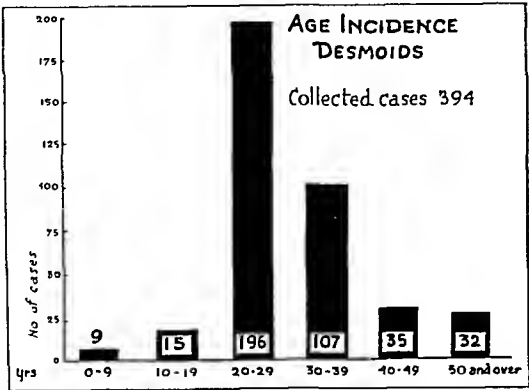

Fig. 1.

prevent recurrence. In the surgical removal a greater or lesser defect is produced in the fascial sheaths which often necessitates a plastic of considerable proportions. In fact, all local tumors of mesodermal origin must be widely excised because recurrence is not uncommon. (Table II.) In our series there was a recurrence rate of 21.7 per cent.
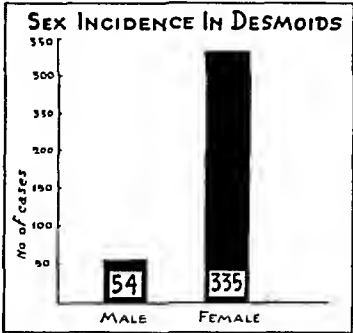
Fig. 2.

Abdominal wall hematomata are particularly difficult to diagnose. They occur most frequently within the rectus sheath. In a review of forty-one cases, Culbertson[10] showed that they usually occurred below and to the left of the umbilicus. They may produce severe or mild symptoms. A hematoma is the one mass in the abdominal wall which may give rise to symptoms of

TABLE I
AGE AND SEX INCIDENCE

	0–10		10–20		20–30		30–40		40–50		Over 50		Unknown	Total
	M	F	M	F	M	F	M	F	M	F	M	F		
Fibroma	1	2	1	3	10	132	4	69	4	10	4	6	34	280
Fibrosarcoma	1	..	..	3	2	21	..	23	2	4	1	3	8	68
Sarcoma	..	3	3	3	5	18	2	7	4	4	3	7	2	61
Myxofibroma	..	..	..	..	..	3	..	1	..	1	..	..	1	6
Myxosarcoma	..	..	..	..	..	...	..	...	..	1	2	..	..	3
Myxoma	..	..	..	..	..	1	..	...	..	..	..	..	..	1
Fibromyxoma	..	..	..	..	..	10	..	6	..	..	..	..	1	17
Fibrolipoma	..	..	..	..	..	..	..	..	..	..	..	2	..	2
Endothelioma	..	..	..	..	..	..	..	..	..	1	..	..	1	2
Angioma	..	..	..	..	..	1	..	..	..	..	..	..	..	1
Angiosarcoma	1	..	..	..	..	..	..	..	..	..	..	..	..	1
	3	5	4	9	17	186	6	106	10	21	10	18	46	442

Whereas only 18.8 per cent of connective tissue neoplasms in all age groups are malignant (Fig. 4), under the age of 20, 48 per cent are malignant. (Fig. 5.) Therefore in the latter age groups thorough in-

such severity as to simulate as acute abdomen. In one of our cases, the hematoma became infected and then, although drained, broke through the peritoneum and the patient rapidly succumbed. In another

case, not reported here, seen by one of us (W. H. M.), the hematoma because of its chronicity was considered to be a malig-
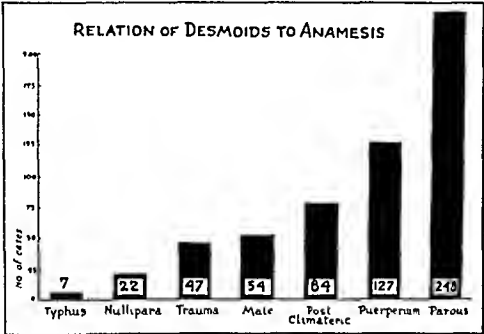

FIG. 3.

nancy either of the stomach or of the transverse colon. It should be emphasized that all cases of hematoma do not necessarily give a positive history of trauma.

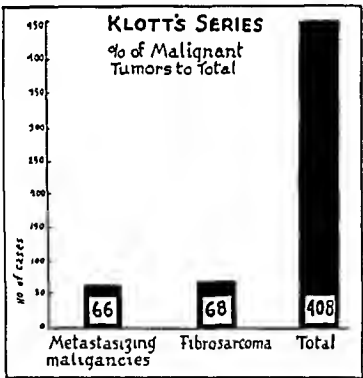Another extremely interesting condition is ossification of the scar of an abdominal

FIG. 4.

wound. The first description that could be found of this condition was by Askanazy[11]

TABLE II

	Pure Fibroma	Cellular Fibroma	Fibro-sarcoma	Sar-coma
Total Cases	92	23	54	44
Local recurrence after radical removal.....	1	1	5	13

in 1900, but it was not until 1909 that a detailed study was made by Lecene.[12] In

296 cases collected by Schulz,[13] 232 gave a history of severe blunt force. Ossification rarely follows in wounds made by sharp
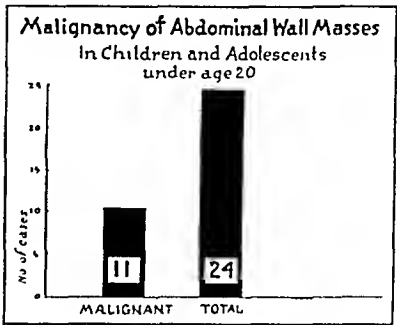
FIG. 5.

instruments. Kuttner[14] in 1910 could find only five cases of ossification of incisional abdominal wounds; in 1925, Bouton reported twenty-five more and added three personal cases. The largest personal, well described series is that of Chauvin and
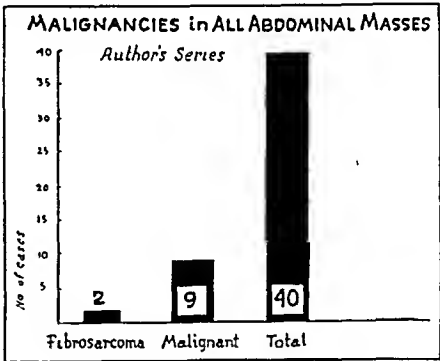
FIG. 6.

Rouslacroix.[16] In these, both direct and chondral bone formation could be demonstrated. The patients, ranging in age from 54 to 74, had cystotomy wounds preparatory to a second stage prostatectomy. At the time of the second operation, the original scars were removed. It is obvious that in this group of cases secondary irritation played an extremely important rôle in this metaplasia.

Calcification and even bone formation may take place in wounds following perfectly clean operations. The former is demonstrated by a case of Dean Lewis:[17]

A white male, age 27, following a gastroenterostomy for an ulcer of the duodenum

developed a painful wound three weeks postoperatively and had at the end of seven weeks a hard mass the length of the wound and a fingerbreadth wide. Roentgenograms showed a marked increase in density.

In Case IV of this series, previously reported in full by I. Cohn,[18] there was undoubtedly histologic evidence of bone formation in a lipoma. In addition calcification may take place in a hematoma. That occasionally a calcified mass may be malignant is demonstrated by a beautifully illustrated case of Pecco's:[19]

A 29 year old white female had a left lower quadrant tumor in the abdominal wall without any symptoms other than an increase in the size of the growth. This was removed and the patient had a rapid recurrence which was removed while she was still in the hospital convalescing from the first operation. The histologic picture was one of new bone formation with numerous osteoblasts. The pathologic diagnosis was osteoblastoma.

Pecco cites a similar case described by Hanseman.

Neurofibromas have been reported in almost every anatomic location in the body and the abdominal wall is no exception. They need not be associated with the typical syndrome of von Recklinghausen's disease. In our series there were three cases (Cases I, III and V) which were characterized by the symptoms of loss of weight, gastrointestinal disturbances, and, in contradistinction to the other primary tumors, by persistent pain.

Along with the other connective tissue tumors must be considered the more common lipoma (Case XXIV) and the infrequent endothelioma and angioma.

Tumors of ectodermal origin occurring in the abdominal parietes are dermoids, moles, and epithelioma. The last occurred usually in the umbilicus and its proximity. One should, however, realize that in this region also occur metastases from intra-abdominal neoplasm, i.e., from the stomach, colon, and ovary (pseudomyxoma peritoni), and in addition, urachal and omphalomesenteric duct epithelial tumors.

Durante[9] described a case of an umbilical tumor in a female 56 years of age, which was present for over twelve years and then suddenly began to grow. When seen it was the size of a mandarin and had ulcerated on the surface. It was excised and showed histologically numerous epithelial pearls and definite signs of malignant changes. He considered this tumor as arising from epithelialized tracts of retracted obliterated vessels and not an urachal tumor.

In our series is a case (Case XXXVI) in which a cyst excised from the lateral abdominal wall could not be histologically differentiated from a sebaceous cyst, but which, however, contained a brownish-black secretion which strongly suggested that it was a dermoid.

Foreign bodies have a rôle in the production of abdominal wall masses. While in the continental literature since the time of Schloffer's[20] report in 1909, there have been sporadic case reports of tumors caused by silk ligatures, there does not seem to be any American or English publications, in spite of the growing use of silk and other non-absorbable sutures and ligature materials. As Marangos[21] has indicated, these tumors may appear from a month to twenty years after the implantation of the foreign body. He cites the following case:

An 18 year old white female had an appendectomy three years before admission to the Clinic. She complained of severe pain, vomited, and was not relieved by conservative treatment. The removal of the tumor promptly relieved the symptoms. Histologic examination of the mass showed it to be fibrous and in the center was a silk ligature.

Characteristically, this type of tumor consists of solid fibrous tissue with an area of round cell infiltration about a foreign body. It is encapsulated but difficult to separate from the surrounding tissue, sharp dissection being required for removal. Schloffer[20] called attention to the fact that the removal of the foreign material usually caused complete regression of the mass. In our series a case of this type (Case II) occurred. A portion of a mass which occurred in a two months old herniorrhaphy

scar ware moved and while the pathologic diagnosis was sarcoma, the remaining tumor disappeared within a month.
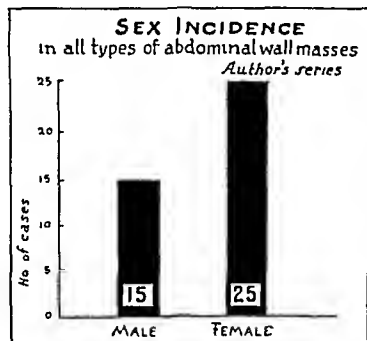

SEX INCIDENCE
in all types of abdominal wall masses
Author's series

FIG. 7.

Recently Feinberg[52] described two cases of abdominal wall granuloma occurring in wounds in which were found talcum powder crystals (magnesium silicate). No doubt these cases should serve to remind the surgeon that a sterile water wash of his gloves before operating may save him and the patient from future trouble.

While there are countries in which irritation of the abdominal wall from hot charcoal pots produces epithelioma, in this country the source of irritation is *x*-ray and radium, which are applied commonly to the lower abdomen of the female. Polano[22] reports such a case as late as 1929:

A white female, age 36, had irradiation for bleeding myoma of the uterus three years before. She was seen in the Clinic with an ulcer of the abdominal wall which had involved the anterior bladder wall. A biopsy showed this to be a squamous carcinoma.

As should be emphasized, irradiation should be given only by those who have a thorough understanding of and a proper training in the physics and administration of *x*-radiation and radium.

Deep abdominal wall abscesses have occurred, some accompanied by severe symptoms and others by mild manifestations. Some follow injury which results in local necrosis, lowered resistance, and then secondary infection; others are bloodborne, such as the typhoid and pneumococcus types. Lavenant[23] reported a case

of contusion hematoma which showed typical signs of inflammation fifteen months after the original injury, without surgical interference in the meantime. In our series is a case (XXVII) following injury, in which the abscess was partially drained and the remainder of the abscess subsequently ruptured intraperitoneally with a resulting fatality. Santoboni[24] reported a case of abdominal wall nonspecific granuloma. A similar case (XXXII) occurred in our series:

A 39 year old female with no history of previous injury had a mass below and to the left of umbilicus. Histologically the tumor was composed of fibrous tissue with focal areas of cellular infiltration consisting of round cells with a few polynuclears. There were no epithelioid or giant cells present which might suggest tuberculosis.

The chronic specific infections, the most important of which are syphilis (gummas) and actinomycosis, offer difficulties in making accurate diagnoses of abdominal wall lesions. In our series there were three gummas (Cases VII, XI, and XXI). One of these was diagnosed as sarcoma and excised. The others were treated conservatively and regressed. Actinomycosis usually is accompanied by secondary sinuses communicating with intraabdominal lesions. However, Saltzmann and Kessler[51] described the following case of actinomycosis, primary in the abdominal wall:

A female, age 18, had a mass the size of a grapefruit in the lower right quadrant which appeared to be fixed to the abdominal wall. There was a small sinus surrounded by induration in the left lower quadrant. Incision of the right lower quadrant mass showed that the sinuses did not penetrate the peritoneum.

Echinococcus cysts[8] have occasionally been reported.

In the present series the male-female ratio is 1 to 1.7 (Fig. 7) which is considerably less than that shown in Klot's series[4]; this comparison shows the fallacy of drawing conclusions from a small number of cases. The incidence of metastatic lesions in abdominal wounds is very small as com-

pared with a large number of abdominal sections done over a period of thirty years.

Practically every intra-abdominal viscus may be the origin for tumors which metastasize to abdominal scars. Wagner[3] reports a papillary cystadenoma of the ovary metastasizing to an abdominal scar and there are other reports of this happening from the same type of tumor (Devorak,[25] Danforth,[26] Tauber,[27] etc.). Gerlack[28] observed extention from a papilloma of the kidney pelvis; Jacobson[29] and Pratt[30] from the endometrium; Mayer[31] from peritoneal carcinomatosis following paracentesis; McDonough[32] from a granulosa cell tumor of the ovary following oöphorectomy, and in our present series implantation in the abdominal wall following surgery of gastric, colonic, and cholecystic carcinoma.

The following are the cases which we have personally seen:

Case 1. E. F., a colored female, entered Charity Hospital in March, 1937, complaining of pain and a "lump" under her ribs on the right side of the abdomen. The pain was colicky in nature and associated with a loss of appetite and vomiting. After a year of such attacks she felt the "lump" beneath the ribs which "worried" her. She had an acute attack in March, 1937 during which she came into the hospital.

Because of the previous attack, the fever, the extreme tenderness, and a mass in the right upper quadrant, the diagnosis of acute empyema of the gall-bladder was made. A laparotomy was performed through a high right rectus incision. There was found a mass of adhesions and omentum over a markedly distended gall-bladder. A trochar was introduced and a pint of pus was evacuated following which a scoop was introduced and over 200 stones removed. A pezzar catheter was inserted into the gall bladder and brought out lateral to the main incision. The patient did fairly well postoperatively and after two weeks an attempt was made to remove the tube but this was unsuccessful. Although every day for ten days further attempts were made, forcible removal of the tube under anesthesia finally had to be done.

The patient returned home and for a period of two months did quite well. She then noticed a steady pain at the site of the drainage wound which had just closed. She tolerated this for eight months, when she noticed a "lump" in the same area.

She reëntered the hospital January 20, 1938. A crust was found over the site of the drainage wound and surrounding it a mass, 4 cm. in diameter, apparently extending to the fascia. The skin showed increased pigmentation and was firmly attached to the tumor. The right rectus wound did not seem to be involved. The patient was obviously in considerable pain. Roentgenologically, the gall-bladder was nonfunctioning. The urine was negative chemically and microscopically. Blood examination showed R.B.C. 2.5, hemoglobin 75 per cent W.B.C. 8,000, with polynuclears 36 per cent, lymphocytes 60 per cent, monocytes 4 per cent. The urea clearance was 100 per cent, the icterus index 8, and hippuric acid liver function test 75 per cent of normal. The Wassermann was negative.

A biopsy showed only scar tissue. It was then decided to remove the mass and possibly do some more radical procedure. However, an incision encircling the tumor showed that it was infiltrating laterally and medially with the main mass about the old drainage tract. A frozen section showed adenocarcinoma, probably from the gall-bladder. No operative procedure other than the removal of the accessible portion of the mass was done. The tumor extended intra-abdominally and there were undoubtedly liver metastases. Roentgenograms of the pelvis, spine, and skull showed no bony metastases. The patient was given deep radiotherapy even though the prognosis was hopeless.

In a careful review of the literature of abdominal wall masses covering the period for Marion Sim's[33] first cholecystostomy in 1878 to the present, we were unable to find a case similar to this, in spite of the fact that cholecystostomy was the most common operation on the gall-bladder until 1920. Of course, it is admitted that carcinoma of the gall-bladder is an uncommon lesion, for, according to Deaver[34] and MacCarty,[35] it occurs but once in 200 operated gall-bladder cases. Deaver states that occlusion of the cystic ducts by malignancy causing stasis results in an

empyema which is commonly the first manifestation of the disease.

CASE II. B. S., white female, age 54, entered Touro Infirmary, January 14, 1930, complaining of "vomiting blood" and coffee ground material. At the onset of the symptoms she had a desire to defecate. She had previously been treated for ulcer over a period of twenty years. Two months before entry to the hospital, the patient had noted a non-painful nodule to the left and above the umbilicus. This was removed and a report of "melanosarcoma" made. The patient left the hospital eleven days later much improved. Following discharge she did not have any more hemorrhages but continued to go downhill and develop masses in the abdomen which were apparently retroperitoneal. The main mass was in the umbilical region and extended into the left lumbar area. However, there were multiple tumors palpable throughout the abdomen and particularly in the iliac gland region. A roentgenogram of the chest was not made. Just before exitus, fluid developed in the abdomen probably from venous obstruction.

As in other locations a mole on the abdominal wall is not necessarily innocent as a wide variety of irritations may occur. However, melanosarcoma does not necessarily have to originate in a nevus because, in the above case and also in Case XXXVI, the lesion started as a subcutaneous nodule and progressed rapidly with fatal termination. Beeker[36] reports a similar case in which the original nodule was on the abdomen and metastases occurred in the axilla.

CASE III. F. T., colored male, age 50, entered Charity Hospital in January, 1938, complaining of two large tumors on his abdomen, loss of weight, and epigastric pain. He had been in the hospital two years before with a similar complaint except that there was but one tumor. On his first admission he was considered to have carcinoma of the stomach with metastases to the region of the umbilicus. He was then discharged and given sedatives for relief of the pain.

Physical examination at the time of his second admission showed an emaciated colored male with two masses on his abdominal wall; one, 1½ inches below the xyphoid, measuring 6 by 8 by 3.5 cm., was covered by skin and was moderately firm in consistency. There were small papillary projections from this mass. The second mass was below and to the left of the umbilicus similar in nature to the first and measured 14 by 12 by 5.5 cm., and on its summit was an ulcer 4 cm. in diameter. There were fewer papillary projections from this one. A biopsy was done as the diagnosis was uncertain. The pathologic report by Dr. Shattenberg was "neurofibroma." The patient refused further therapy and deserted from the hospital.

CASE IV. E. L., colored female, age 55, was admitted to Charity hospital September 18, 1934, complaining of "hernia" in the right lower abdomen and high blood pressure. From birth, the patient had had a soft mass in the right lower abdomen which she claimed was reducible before puberty but not since then. It had remained constant in size since puberty.

Physical examination showed a mass in the right lower abdomen extending from the anterior superior spine to beyond the symphysis pubis. It measured 16 by 8 by 8 cms. The skin was smooth, the veins slightly prominent, and when the skin was stretched there was some dimpling. The mass was lobulated and in its lower half some hard, somewhat sharp projections could be felt. It transmitted light in most of its extent. It could not be reduced. There were no enlarged inguinal nodes. Examination by x-ray showed an irregular calcified mass, probably a cyst to the right of the midline and apparently in the pelvis. The mass was excised and found to be a partially ossified lipoma. A roentgenogram of the pelvis after convalescence showed the previously mentioned calcified mass still present. Bimanual examination identified this as a calcified ovary.

COMMENT

The diagnosis of abdominal wall masses is not difficult if they are kept in mind. Their differentiation from intra-abdominal tumors is of prime consideration. If they are fairly well limited, it is relatively easy to get the "feel" of the mass as being either intra-abdominal or intramural. By far the greatest number of these masses occur below the umbilicus and in the female are most frequently desmoids. However, hematomata have a tendency to blend with the

surrounding tissues and are therefore most confused with intra-abdominal lesions. Culbertson[10] showed that abdominal wall lesions were suspected and diagnosed only five times in forty-one cases, intra-abdominal conditions being diagnosed in all of the others. Fothergill[37] and Gellert[38] had similar experiences. Halban[39] suggests bimanual examination with the vaginal hand touching the lower pole of the tumor which, if intramural, recedes from the hand when the patient sits up and at the same time appears more prominent on the wall as the muscles contract. If the mass is in the abdomen the reverse is true. In the male, the same methods may be used using the rectal route. With the recent perfection of abdominal endoscopy there is no good reason why peritoneoscopy should not be used in differentiating parietal from visceral masses. We believe that an x-ray examination of the chest and skeleton for possible metastases should always be done before treatment is instituted.

It may well be said that the most practical method of diagnosis is biopsy followed by microscopic examination of the tissues.

The prognosis varies with the histology and location of the tumor, the completeness of operative removal and the presence or absence of metastases. We believe that the presence of malignancy in a scar does not necessarily indicate that the intra-abdominal lesion has progressed. It may be only a local recurrence, probably due to a break in the operative technique or the deliberate failure properly to protect the abdominal wall at the time of the original procedure. We are aware of the difference of the biologic response in each patient to the new growth and therefore there are times when only the continued follow-up of the patient will give the final answer.

The following thirty-six cases were abstracted from the available records of Charity Hospital for the years 1906 to 1939, and together with the cases reported in detail, form the basis for the authors' statistics.

I. NEUROFIBROMA: (1906) A colored male, age 33, was hospitalized in March, 1906, complaining of sore spot at the umbilicus associated with swelling which began early in 1905. A rapid increase occurred in size of the tumor during March, 1906, with marked loss of weight and intermittent darting pain about the tumor mass. The patient was emaciated. A large tumor was present at the umbilicus with small nodules on it. It was movable with the abdominal wall and not attached to intra-abdominal structures. Clinical diagnosis was fibrosarcoma. Treatment: excision. Pathologic diagnosis: neurofibroma. Result: recovered but no follow-up.

II. MYXOSARCOMA: (1911) A white male, age 52, had a herniorrhaphy two months before. Two weeks after leaving the hospital he noticed a lump under the scar, which was not painful but was tender. The patient was well nourished. A firm mass, the size of a large pecan was apparently fixed in the scar just above the right inguinal ligament. Clinical diagnosis: none. Pathologic diagnosis: myxosarcoma. Treatment: partial excision. Result: cured, no recurrence.

III. FIBROMA: (1911) A white male, age 16, had been struck in the lower abdomen three months before. He developed a mass below the umbilicus over a period of two weeks. No further increase in size occurred. Well nourished. There was a mass 2.5 inches in diameter, below and to the left of the umbilicus, apparently fixed in the wall, possibly rectus sheath. Clinical diagnosis: sarcoma. Treatment: excision. Pathologic diagnosis: fibroma with hemosiderin. Result: cured.

IV. FIBROSARCOMA: (1912) A white female, age 22, noticed a small lump six months before admission. It grew slowly and there were no other symptoms. There was a firm mass beneath the skin in the region of McBurney's point. The skin moved freely over it. Clinical diagnosis: ab-

dominal wall tumor. Treatment: excision. Pathologic diagnosis: fibrosarcoma. Result: no recurrence.

V. NEUROFIBROMA: (1912) A colored male, age 56, complained of a tumor in the right upper abdomen which had grown rapidly over a period of six weeks and was accompanied by moderate persistent pain. There was a mass the size of an orange beneath the skin and attached to the underlying muscles and fascia in the right upper quadrant. Clinical diagnosis: abdominal wall tumor. Treatment: excision. Pathologic diagnosis: neurofibroma. Result: unknown.

VI. FIBROMA: (1914) A colored female, age 45, had a tumor removed from the belly wall ten months before. Two months later she noted a "knot" in the scar. There was no pain, but there had been hemorrhage from the vagina for one month. The tumor was in the midline below the umbilicus and was attached to the scar. It was the size of an orange, non-tender, and the skin moved over it except at the scar. Clinical diagnosis: sarcoma. Treatment: excision. Pathologic diagnosis: fibroma (desmoid). Result: unknown.

VII. GUMMA: (1915) A white female, age 34, complained of pain in the epigastrium to the left of the midline, present for six months. There was occasional relief for periods of two to three days. For two months she had noticed a tumor. This thin female had a hard, non-tender, midline supra-umbilical mass, poorly delimited, which seemed to be in abdominal wall. Wassermann reaction was 4 plus. Clinical diagnosis: sarcoma. Treatment: excision with iodides and mercury following surgery. Pathologic diagnosis: gumma. Result: unknown.

VIII. FIBROMA: (1915) A colored female, age 36, had noted a pimple on the skin of the abdomen one year before admission. This increased in size until three weeks before admission, when it burst. White creamy material was discharged and a decrease in size followed. The patient had lost 15 pounds. She was undernourished, with a mass 3 inches in diameter, definitely limited, the bulk of which seemed to be in the abdominal wall. The skin was adherent only at the ulcerated area. Wassermann negative. Clinical diagnosis: gumma. Treatment: iodides and mercury without result. Excision with removal of half of rectus muscle. Pathologic diagnosis: fibroma (desmoid) with areas of infections and necrosis. Result: cured, no recurrence.

IX. FIBROMA: (1916) A colored female, age 19, noted a lump in the "stomach" ten months before admission. There was gradual increase in size, but no pain until four days before entry. The abdomen was lax, with a tumor in the left side, the size of a small pumpkin. It was apparently attached to the left rectus muscle. Wassermann positive. Clinical diagnosis: gumma. Treatment: iodides and mercury for two months without result, followed by biopsy. Pathologic diagnosis: fibroma (desmoid). Result: unknown.

X. FIBROSARCOMA: (1916) A colored female, age 35, noticed a knot in the right side of the abdomen ten months before admission, which steadily increased in size. A biopsy was done elsewhere, but the patient did not come into the hospital until five months after. A mass 3 inches in diameter was seen in the abdominal wall. A bleeding ulcer was on its summit. Wassermann negative. Clinical diagnosis: benign abdominal wall tumor. Treatment: excision. Pathologic diagnosis: fibrosarcoma. Result: three recurrences in thirty months.

XI. GUMMA: (1920) A colored male, age 41, complained of a painless tumor in mid-abdomen, present for five months. A similar one appeared two months after the other, but gradually disappeared over a period of six weeks. The patient was thin with a raised, flat, non-tender tumor 2.5 inches in diameter, with indefinite

border in the abdominal wall above and to the left of the umbilicus. There was no bulge on coughing and the rectus sheath seemed involved. Wassermann positive. Clinical diagnosis: gumma. Treatment: iodides and mercury rubs. Result: tumor disappeared in seven weeks.

XII. FIBROMA: A colored female, age 22, had noted a tumor, the size of a marble in the right lower abdomen one year before admission. It was not painful at first but seven months later it became so. A miscarriage occurred six weeks before entry to hospital. The patient was well nourished and the abdominal wall lax. In the right lower quadrant, just above the pubic spine was a hard, irregular movable mass, size of baseball. It was palpable through the vagina, but it was difficult to determine if it was of adnexal origin. Wassermann negative. Clinical diagnosis: intraligamentary myoma. Treatment: excision of mass and portions of external oblique, internal oblique and transversalis muscles. Peritoneal cavity entered. Pathologic diagnosis: fibroma (desmoid). Result: cured, no recurrence.

XIII. FIBROMA: (1922) A colored female, age 40, complained of a lump in the abdomen, which began three months after a gall-bladder operation (August, 1919). Some drainage came from the wound at intervals since then. The mass slowly increased in size. The patient was obese. A draining sinus was seen in the old scar. To the right of the sinus and umbilicus was a firm mass, the size of an orange, which was freely movable laterally but not caudad or cephalad. The overlying skin was warmer than the surrounding skin, and movable over the mass, which was tender on pressure. Wassermann not reported. Clinical diagnosis: abscess. Treatment: incision followed by excision. Pathologic diagnosis: fibroma (desmoid). Result: cured, no recurrence.

XIV. FIBROMA: (1924) A colored female, age 20, four months before entry noticed a lump on her abdomen inside her hip bone. The tumor gradually increased in size until she was unable to put her clothes over it. A swelling just medial to the anterior superior spine of the iliac crest, the size of an orange, movable and very firm, was apparently attached to ilium. Wassermann not reported. Clinical diagnosis: sarcoma of ilium. Treatment: excision of mass along with portion of external oblique, internal oblique and transversalis muscles. Pathologic diagnosis: fibroma (desmoid). Result: recovered, but no follow-up.

XV. ROUND CELL SARCOMA: (1925) A colored male, age 27, had a gunshot wound of the abdomen ten months before entry, followed by sinus formation which healed and recurred. Two months before entry he noted a small lump about sinus. Patient had lost 35 pounds and showed marked loss of weight. Numerous acorn-sized masses were beneath the skin of the abdomen. Four scars were present, one draining. No enlarged lymph nodes were noted. Wassermann negative. Clinical diagnosis: abdominal sinus. Multiple tumors. Treatment: biopsy. Pathologic diagnosis: round cell sarcoma. Result: died, no autopsy.

XVI. METASTASIS FROM EMBRYOMA OF TESTIS: (1925) A colored male, age 65, four months before entry noticed a growth on the abdomen just below the navel. One month later he noticed a lump in the groin, followed by many smaller ones on the abdomen. He was elderly and thin. Below the umbilicus were many rounded masses varying from peanut to golf ball size. The overlying skin was purplish pink and bled easily when irritated. Genitourinary examinations were negative. The extremities showed pitting edema. Clinical examination: multiple abdominal wall tumors. Treatment: none. Pathologic diagnosis (autopsy): embryoma of testis with metastases to

inguinal nodes, abdominal wall and diaphragm.

XVII. FIBROMA: (1925) A white female, age 27, noticed a lump next to the navel, six months before entry, which increased in size rapidly and was painful on exertion, such as coughing, sneezing, and defecation. Below and to the right of the umbilicus was a firm, tender tumor mass size of hen's egg which was unreducible and did not transmit impulse on coughing. Clinical diagnosis: lipoma. Treatment: excision with portion of rectus sheath. Pathologic diagnosis: fibroma (desmoid). Result: cured, no recurrence.

XVIII. FIBROMA: (1925) A colored female, age 27, noticed a "knot" in the right side of her abdomen one year before entry. It grew from marble size to fist size in three months and was excised elsewhere. Six months after surgery the "knot" appeared in the scar and grew very rapidly. On the abdomen, underlying a scar and extending from the costal margin to just above the symphysis was a multilobular freely movable mass, 8 by 4 by 5 inches, which varied in consistency, soft in some areas and hard in others. Clinical diagnosis sarcoma. Treatment: excision of mass and four-fifths of right rectus sheath and muscle. Pathologic diagnosis: fibroma (desmoid). Result: cured, no recurrence.

XIX. FIBROMA: (1929) A white male, age 43, noticed a lump on the abdomen four months before entry. The lump was painful at intervals. There was a marble-sized mass to the left of the umbilicus, palpable but not visible. No cough impulse was present and the skin was freely movable over the mass. Wassermann negative. Clinical diagnosis: fibroma. Treatment: excision along with portion of rectus sheath and muscle. Pathologic diagnosis: fibroma (desmoid). Result: no follow-up.

XX. GUMMA: (1930) A colored female, age 35, noticed a hard lump in the right side of the abdomen above the navel two weeks before admission. The tumor was not painful. The patient was thin, not acutely ill. There was a rounded mass in the upper right abdomen beneath the skin. The skin was tender and slightly warmer than the surrounding integument. Generalized lymphadenopathy, temperature 98.6°F., Wassermann positive. Clinical diagnosis: gumma. Treatment: iodides and mercury. Results: mass disappeared by thirty-seventh day.

XXI. FIBROMA: (1930) A white female, age 38, was operated on two years before entry for a tumor on the abdomen. Four months after surgery some pain was felt in the scar and then the tumor was noted. There was a mass in the right upper quadrant, 2 inches in diameter and beneath old scar. It lay just below the rib and seemed attached to it. Wassermann negative. Clinical diagnosis: chondroma. Treatment: excision. Pathologic diagnosis: fibroma (desmoid). Result: no recurrence.

XXII. FIBROMA: (1931) A white female, age 23, had intermittent pain below the umbilicus for six months postpartum. Two years later she noticed a tumor just above the pubic bone, in which pain occurred on exertion and coughing. In the lower abdomen just above symphysis pubis was a smooth globular mass 6 inches in diameter, firm and attached to pubis. The overlying skin was not attached. The mass was moderately tender Wassermann negative. Clinical diagnosis: fibrosarcoma. Treatment: excision of mass which was found to involve rectus sheath, conjoined tendon, fascia of external oblique, and internal oblique and transversalis muscles. Pathologic diagnosis: fibroma (desmoid). Result: no recurrence and no hernia.

XXIII. FIBROLIPOMA: (1932) A colored female, age 50, noticed a lump the size of a marble in the left lower abdomen two months before entry. The mass grew to the size of a grapefruit in two months. One week before entry

the tumor became painful. The patient was obese. There was a mass, in the left lower quadrant just above and medial to Poupart's ligament. The tumor was fixed, non-tender and the overlying skin shiny. Wassermann positive. Clinical diagnosis: sarcoma? gumma? Treatment: excision of encapsulated mass easily peeled from external oblique fascia. Pathologic diagnosis: fibrolipoma. Result: cured.

XXIV. HEMATOMA: (1933) A colored female, age 49, complained of pain in the abdominal tumor which she noticed eight days before entry. The mass appeared about six hours after she had lifted a heavy clothes tub. There was a mass in the right lumbar region 2 inches in diameter. The overlying skin was slightly discolored and the mass acutely tender. Wassermann negative. Clinical diagnosis: hematoma. Treatment: aspiration of 5 c.c. of old blood with slight reduction in size of tumor and relief of pain. Result: mass disappeared in forty days.

XXV. FIBROMA: (1934) A colored female, age 21, complained of pain in the lower right side of two years' duration, and for two months before entry she was unable to lie on that side. The patient had not noticed any tumor. There was a fairly firm, orange-sized mass in the right lower quadrant, which felt cystic, was movable, and vaginal examination suggested that it was attached to adnexa. Wassermann negative. Clinical diagnosis: ovarian cyst. Treatment: laparotomy showed mass anterior to peritoneum and involving lateral border of rectus sheath, conjoined tendon, external oblique fascia, internal oblique and transversalis muscles. Excision of mass. Pathologic diagnosis: fibroma (desmoid). Result: recovered, but no follow-up.

XXVI. INFECTED HEMATOMA: (1934) A colored female, age 41, was struck in the upper abdomen by a large stone three weeks previous to admission. This injury was followed by a moderate sticking pain and a week later the patient noticed an egg-sized mass in the upper right abdomen at the site of injury. Fever was present for a week. The patient was well nourished and moderately ill. A circumscribed, tender, slightly movable mass extended from below the ribs to just below the umbilicus on the right. The skin was movable over the mass and there was no increased heat. Temperature was 103°F. Wassermann negative. Clinical diagnosis: intraperitoneal hematoma, secondary infection with localization? Liver abscess? Treatment: incision and drainage of extraperitoneal abscess. Result: died following rupture of abscess pocket into peritoneal cavity.

XXVII. METASTATIC CARCINOMA: A colored male, age 52, had partial gastrectomy for carcinoma of stomach ten months before entry. Two weeks before admission began to vomit and have pain in the scar. The patient was emaciated. There was an indefinite mass in the abdomen and multiple nodules in and about the scar. Wassermann negative. Clinical diagnosis: metastatic carcinoma from stomach. Treatment: biopsy. Pathologic diagnosis: adenocarcinoma. Result: died.

XXVIII. ACUTE AND CHRONIC PHLEGMON: (1935) A white male, age 45, had a sudden sharp pain in the lower abdomen three weeks before entry. The pain gradually disappeared over period of three days. The patient then noticed a hard tumor in the right lower part of the abdomen. There was a hard, walnut-sized, slightly tender tumor with boundaries fading out into the surrounding tissue. It was apparently fixed in the muscles. Clinical diagnosis: abdominal wall tumor. Treatment: a tumor involving the external oblique, internal oblique transversalis muscles and transversalis fascia was excised. The peritoneal cavity was opened and the omentum found adherent to peritoneum at tumor site. Patho-

logic diagnosis: phlegmonous infiltration with large number of eosinophils and multiple areas of necrosis. Result: unknown.

XXIX. FIBROMA: A colored female, age 24, fell during pregnancy three years before entry. Shortly after the injury the patient noticed a tumor in the left side which did not bother her until three weeks before admission when pain occurred with coughing or laughing. There was an orange-sized, firm mass in the left upper quadrant although movable seemed attached to the wall rather than to intra-abdominal structures. Wassermann *positive. Clinical diagnosis: gumma?* rectus sheath tumor? Treatment: excision of mass along with part of rectus muscle and sheath. Pathologic diagnosis: fibroma (desmoid). Result: no recurrence or hernia.

XXX. FIBROMA: (1936) A colored female, age 27, noticed an egg-sized lump sixteen months before entry. The tumor gradually increased in size. Last menstrual period two months before admission. There was a mass 15 cm. in diameter extending from the umbilicus to just above *symphysis pubis and a little to the left.* The mass was smooth, firm and the skin moved easily over it. Wassermann negative, temperature 99°F. Clinical diagnosis: umbilical tumor, pregnancy. Treatment: excision of mass with portion of both rectus muscles and sheaths. Pathologic diagnosis: fibroma (desmoid). Result: no recurrence, large hernia.

XXXI. SCHLOFFER TUMOR: (1936) A white female, age 39, had pelvic operation four months before entry. Two weeks after surgery a lump was noticed in *upper end of scar. One week before* admission became painful and a small abscess drained leaving a hard lump beneath scar. There was a small granulating area in the upper end of the laparotomy scar with a golf ball-sized hard mass beneath, apparently extra-peritoneal. Clinical diagnosis: ulcerating malignancy. Treatment: excision of tumor by sharp dissection as no line of cleavage was present. Pathologic diagnosis: chronic granulation tissue with marked fibroblastic proliferation (Schloffer's tumor). Result: cured.

XXXII. METASTATIC CARCINOMA: (1936) A white male, age 52, had carcinoma of the descending colon removed in December, 1934. In January, 1936 a nodule, which had been present two months, was removed from the scar. The patient returned in May, 1936 complaining of pain and tumor in the scar. He was fairly well nourished. There was a hard mass 1.5 *inches in diameter in the scar and* firmly attached to the skin and underlying scar tissues. Wassermann negative. Clinical diagnosis: metastatic carcinoma from colon. Treatment: wide excision. Pathologic diagnosis: adenocarcinoma. Result: no recurrence for two years, no follow-up since.

XXXIII. DERMOID: A colored male, age 22, noticed at age 10 a small painless lump in the left side of the abdomen which had grown slowly and at intervals. There was a soft, goose egg-sized, freely movable mass above the left anterior superior iliac spine. Wassermann negative. Clinical diagnosis: lipoma. Treatment: excision of mass superficial to external oblique muscle. Pathologic diagnosis: dermoid contained thick brownish black fluid. Result: cured.

XXXIV. SPINDLE CELL SARCOMA: (1937) A colored male, age 17, noticed a painless lump in the right lower abdomen three months before admission. The mass increased in size rapidly. There was a large, nodular, slightly tender mass in the right lower quadrant extending to the pubic region and displacing the penis to the left. Wassermann negative. Clinical diagnosis: sarcoma. Treatment: excision of tumor. Pathologic diagnosis: spindle cell sarcoma. Result: pulmonary metastases and death in four months.

XXXV. SPINDLE CELL SARCOMA: (1937) A white male, age 52, had tumor removed from abdomen 30 months before admission. Eighteen months before entry he noticed a growth in the scar, which had grown slowly. There was a firm, non-tender, baseball-sized tumor in the old scar, which was firmly attached to the skin and underlying structures. Wassermann negative. Clinical diagnosis: abdominal wall tumor. Treatment: excision, removed scar, muscle and fascia down to peritoneum. Pathologic diagnosis: highly malignant spindle cell sarcoma. Result: no recurrence in a year, no evidence of metastases elsewhere.

XXXVI. FIBROMA: (1937) A colored female, age 26, noted a small mass below and to the right of the navel four months before admission. It was not painful and grew slowly. The last menstruation had occurred five months before. There was a rounded, firm, delimited, non-tender mass, the size of a large lemon, to the right and just below umbilicus. The overlying skin was normal and moved freely over the mass which was apparently superficial. No cough impulse present. Wassermann negative. Clinical diagnosis: fibroma, pregnancy. Treatment: excision of tumor with portions of rectus sheath. Pathologic diagnosis: fibroma (desmoid). Result: no recurrence or hernia at time of delivery four months after surgery.

SUMMARY

1. An orderly classification of abdominal wall masses is proposed.

2. The incidence of abdominal wall lesions as regards age and sex, relative malignancy and relation to previous history of the patient is given.

3. The frequency of desmoids in the female and high ratio of malignant to non-malignant masses in children and adolescence are emphasized.

4. Schloffer's foreign body (silk) tumor is either not being recognized or else not reported in the English speaking countries.

5. Abdominal wall hematomas should be strongly considered in the differential diagnosis of abdominal masses.

6. Forty-one cases of abdominal wall masses are reported from Charity Hospital and Touro Infirmary, New Orleans. Of these an adenocarcinoma of the gallbladder metastasizing by direct extention to a cholecystostomy wound is reported for the first time.

REFERENCES

1. MACFARLANE, J. Clinical reports of the surgical practise of the Glasgow Royal Infirmary, Glasgow 63 and 66, 1832.
2. MULLER, J. Uber den feineren Bau und die Formen der Krankhaften Geschwulste, Berlin, 1: 60, 1838.
3. WAGNER, E. Fall von mehrfachen Cysten des Ovariums. Peritonaeums und des subcutanen Bindegewebes. Arch. f. Heilk., 92, 1864.
4. KLOT, B. Bauchdeckentumoren: 408 cases. Beitr. z. klin. Chir., 123: 1, 1921.
5. PFEIFFER, C. Die desmoids der bauchdecken und ihre Prog. Beitr. z. klin. Chir., 44: 334, 1904.
6. LEDDERHOSE, G. Deutsche Ztschr. f. Chir., 45, 1897. (Quoted by Klot.[4])
7. GURLTS. Quoted by Zweifel and Payr. Die Klinik der boesartigen geschwulste unter Mitwirkung von Boeminghaus. Max Borst (et al.) Leipzig, 1924.
8. LENORMANT, C. Les kystes hydatiques de a paroi abdominale. Rev. de Chir., 32: 446, 1905.
9. DURANTE, G., and ROULLAND, H. Epithelioma of umbilical origin. Gynecologie, 28: 65, 1929.
10. CULBERTSON, C. Hematoma occurring spontaneously in sheath of rectus abdominus muscle. J. A. M. A., 85: 1955, 1925.
11. ASKANAZY. Verhandlung der deutsch. pathol. Gesellschaft. Aachen, p. 106, 1900.
12. LECENE, P. Ossification dans une citatrice de laparotomie. Bull. et mém. Soc. anat. de Paris, 84: 279, 1909.
13. SCHULZ. Quoted by Lewis, D.[17]
14. KÜTTNER, R. Myositis ossificans circumscripta. Ergeb. d. Chir. u. Orthop., Bd. 1.
15. BOUTON, M. L'ossification hétérotypique dans les cicatrices opératoires. Thèse de Paris, No. 543.
16. CHAUVIN, E., and ROUSLACROIX, A. Formation of bone in laparotomy scars. J. d'urol., 27: 465, 1929.
17. LEWIS, DEAN. Myositis ossificans developing in a clean incised abdominal wound. S. Clinics Chicago, 1: 1119, 1917.
18. COHN, I. Masses in the groin. Internat. Clin., 2: 229, 1935.
19. PECCO, R. Osteoblastoma of the abdominal wall. Case. Arch. ital. de chir., 30: 441, 1931.
20. SCHLOFFER, H. Ueber chronisch-entz undliche Bauchdengeschwulste nach Buchoperationem. Verb. d. deutsch. Gesellsch. f. Chir., 37: 582, 1908.

21. Marangos, G. Chronic inflammatory tumors of the abdominal wall (Schloffer's). *Wien. klin. Wchnschr.*, 47: 202, 1934.

22. Polano, O. Roentgen carcinoma of the abdominal wall. *Zentralbl. f. Gynäk.*, 53: 1426, 1929.

23. Lavenant, A. Phlegmon of the abdominal wall fifteen months after contusion. *J. d'urol.*, 27: 465, 1929.

24. Santoboni, U. Nonspecific granuloma. *Minerva Med.*, 2: 535, 1932.

25. Dvorak, H. Implantation malignant of abdominal wall. *Surg., Gynec. & Obst.*, 50: 907, 1930.

26. Danforth, W. C. New growths after laparotomy. *Surg., Gynec. & Obst.*, 29: 175, 1919.

27. Tauber, R. Malignant degeneration after removal of papillary ovaries. *Zentralbl. f. Gynäk.*, 51: 1505, 1927.

28. Gerlack, K. Papillary tumors of the renal pelvis. *Deutsche Ztschr. f. Chir.*, 239: 750, 1933.

29. Jacobson, V. C. Intraperitoneal transplants of endometrial tissue. *Arch. Path. & Lab. Med.*, 1: 169, 1926.

30. Pratt, J. P. Adenomyoma of endometrial implants in the abdominal wall. *J. Michigan State M. S.*, 26: 82, 1927.

31. Mayer, M. Implantation of metastases in the abdominal wall. *München. med. Wchnschr.*, 59: 1439, 1912.

32. McDonough, W. B. P. Granulosa cell tumor metastases to the abdominal wall. *Am. J. Obst. & Gynec.*, 33: 657, 1937.

33. Sims, M. Quoted from Keen, W. W. and White, J. W. American Textbook of Surgery, p. 748. Philadelphia, 1893. W. B. Saunders Co.

34. Deaver, J. B. Carcinoma of the gall-bladder. *M. Rec.*, 96: 47, 1919.

35. MacCarty, W. C. Gall-bladder disease. In Dean Lewis' Surgery Vol. 7, Chapter 11.

36. Becker, J. Melanosarcoma of abdominal wall. *Arch. f. klin. Chir.*, 187: 530, 1936.

37. Fothergill, W. E. Hematoma in abdominal wall simulating pelvic new growth. *Brit. M. J.*, 1: 941, 1926.

38. Gellert, H. H. Hematoma of the abdominal wall. *Brit. M. J.*, 2: 12, 1926.

39. Halban, J. Differential diagnosis of abdominal wall tumors. *Wien. med. Wchnschr.*, 85: 790, 1935.

40. Nichols, R. W. Desmoid tumors. *Arch. Surg.*, 7: 227, 1923.

41. Schuman, E. D. Series of cases presenting abdominal tumors of unusual type which give rise to difficulties in diagnosis. *S. Clin. North America*, 4: 185, 1924.

42. Donald, J. S., and Caylor, H. D. Sarcomatous degeneration of a desmoid. *Ann. Surg.*, 89: 631, 1929.

43. Straub, G. L. Desmoid. *California & West. Med.*, 31: 186, 1929.

44. Bessesan, D. H. Desmoid tumor of abdominal wall. *Am. J. Surg.*, 16: 513, 1932.

45. Crossan, E. T. Desmoid. *S. Clin. North America*, 9: 1245, 1929.

46. Walters, W., and Church, G. Desmoid tumors of left rectus muscle. *S. Clin. North America*, 14: 647, 1934.

47. Marlow, F. W. Desmoids. *Canad. M. A. J.*, 32: 674, 1935.

48. Gutierrez, A. Desmoids. *Bol. y trat. Soc. de cir. Buenos Aires*, 19: 830, 1935.

49. de Pol, G. I tumori connettivali degli strati muscoloapo-neurotici delle pareti abdominali. *Policlinico (sez. prat.)*, 43: 1067, 1936.

50. Penick, R. Desmoids occurring in operative scars. *Internat. S. Digest.*, 23: 323, 1937.

51. Saltzman, H. A., and Kessler, I. *Arch. Dermat. & Syph.*, 36: 131, 1937.

52. Fienberg, R. Talcum powder granuloma. *Arch. Path.*, 24: 36, 1937.

THE PREOPERATIVE AND POSTOPERATIVE CARE OF PATIENTS WITH GASTRIC CANCER*

GEORGE T. PACK, M.D.   AND   ISABEL M. SCHARNAGEL, M.D.

Attending Surgeon, Memorial Hospital

Assistant Attending Surgeon, New York
Infirmary for Women and Children

NEW YORK CITY

LORD Moynihan has said: "Surgery has been made safer for the patient; we must now make the patient safe for surgery." Twenty-five years ago George Grey Turner made the statement that those who contemplated operation for gastric cancer had "no thought but to kill two at a blow"—the patient and their own reputations. However, the present success attending the surgical treatment of gastric cancer is due not only to improvement in operative technique but also to a better knowledge of the preoperative and postoperative care of the patient.

PREOPERATIVE CARE

There are several consequences of gastric cancer one or more of which are nearly always present and must be considered in the preoperative preparation. These complicating conditions are: dehydration, malnutrition, anemia, gastric dilatation and retention due to pyloric obstruction.

Dehydration and Malnutrition. Dehydration and malnutrition are usually present in candidates for gastric surgery due to vomiting or abstinence from food and fluids for fear of pain or discomfort. Fluids given by mouth are preferable if they can be retained. Supplementary administration of fluids by parenteral routes is described on page 442.

In establishing a high glycogen reserve, ingested glucose has been shown to give a higher and longer sustained blood sugar elevation than glucose administered by the intravenous route. Sugar may be given as honey, lactose in orange juice, or in other ways up to the night before operation. Seale Harris employs lemonade containing 1 ounce of strained lemon juice to 5 ounces of 10 per cent glucose solution; or 2 ounces of strained orange juice to 4 ounces of glucose solution. These preparations may be given in 6 ounce feedings every hour during the day and every two or three hours at night when the patient is awake, up to within six hours of the operation. It can be used in the presence of cancer causing moderate obstruction.

No protein food should be given for twenty-four hours before operation, because water passes slowly out of the stomach during gastric digestion. Food containing protein would therefore favor or increase gastric retention. Fats are retained in the stomach even longer than protein and must be avoided in patients with gastric retention. Fats in excess may add to the acidosis associated with the operation.

During the preoperative administration of fluids, consideration should be given to the large percentage of patients with cancer of the stomach who have achlorhydria or low gastric acidity. Horsley keeps $\frac{1}{2}$ of 1 per cent solution of hydrochloric acid sweetened to taste in a pitcher by the patient's bed for two days before operation and encourages its ingestion at frequent intervals. The antiseptic value of the hydrochloric acid is an additional advantage. Thirty to 60 minims of dilute hydrochloric acid may also be given in water three times daily, with pepsin gr. 2 and pancreatin gr. 2 to aid digestion.

Anemia. Gross hemorrhage from gastric cancer is not common, but long continued imperceptible small hemorrhages are the rule. Severe anemia is usually associated

with gastric cancer, particularly the polypoid tumors situated along the greater curvature and in the fundus. This anemia has been confused on occasions with primary or pernicious anemia when only the changes in the cells of the circulating blood were considered (Doan). Because of the similarity of the blood picture to pernicious anemia and the known relation between the mucosal functions of the stomach and hematopoiesis, it is logical to assume that changes in this mucosal function may play a part in the causation of the anemia of gastric cancer.

Castle believes that the nutritional defect in patients with pernicious anemia is caused by failure of a reaction which occurs in the individual between food (the extrinsic factor) and a substance in the normal gastric secretion (the intrinsic factor). The administration of either factor separately has no effect (i.e., beef muscle—normal human gastric juice) but when they are given together (i.e., contact before or after administration to the patient) an improvement is seen within ten days and it is progressive. The activity of desiccated hog stomach mucosa is due to the presence of both intrinsic and extrinsic factors and positive effects are obtained from 30 Gm. daily. Castle and Ham represent the factors in a schematic formula: $\dfrac{F \times G}{I} = L.E.$ F is the food (extrinsic) factor; G is the gastric (intrinsic) factor; I represents intestinal impermeability and $L.E.$ liver extract. Probably in none of the macrocytic anemias (as pernicious anemia, sprue, *gastric cancer*, dibothriocephalus latus infestation) is any factor on the left of the equation completely normal.

This knowledge is most helpful in the postoperative treatment of the anemia that may follow total or subtotal gastrectomy. The administration of such hematopoietic agents as desiccated gastric mucosa and liver extract is usually reserved for the postoperative state. The first step in the treatment of the anemia of gastric cancer is to remove the source of bleeding by

gastrectomy and it is unwise to delay operation while attempting to improve the blood by medical means.

If the hemoglobin is below 40 per cent, blood transfusions are employed preoperatively and if response can be obtained without too much delay, an increase to 60 per cent is desirable. Even if there is little response the blood is of value and donors should be ready for use immediately after the operation.

Relief of Dilatation Due to Pyloric Obstruction. The oral administration of fluids and food may be impossible due to retention with dilatation of the stomach by partial or complete pyloric obstruction. This obstruction usually develops slowly in cancers of the distal third of the stomach and if accompanied by long continued vomiting is associated with disturbances in the acid-base balance or electrolytes of the blood. Gastric tetany following pyloric obstruction occurs when the cause of the obstruction is inflammatory as in infants, or adults with fibrosing ulcers and hyperchlorhydria, but is not a problem in the case of gastric cancer. Nevertheless, any pyloric obstruction, neoplastic or otherwise, which causes persistent vomiting will increase the carbon dioxide combining power of the blood and furnish an indication for the parenteral administration of chlorides.

A part or all the fluids must then be given by hypodermoclysis or intravenous infusions, preferably the latter method. The chemical status of the blood should be checked every forty-eight hours and normal saline or Ringer's solution given when necessary to maintain the level of blood chlorides.

The mucosa of the non-cancerous segment of the stomach is usually infected, edematous and congested; the gastric wall is atonic. These tissues are difficult to suture and, moreover, constitute a source of danger from infection of the suture line and possible perforation. Repeated gastric lavage and rest of the stomach with improvement of the general condition of

the patient by intravenous fluids will lessen the edema and congestion of the mucosa.

one or two days before operation and the stomach lavaged with hydrochloric acid solution especially if the free acidity is low
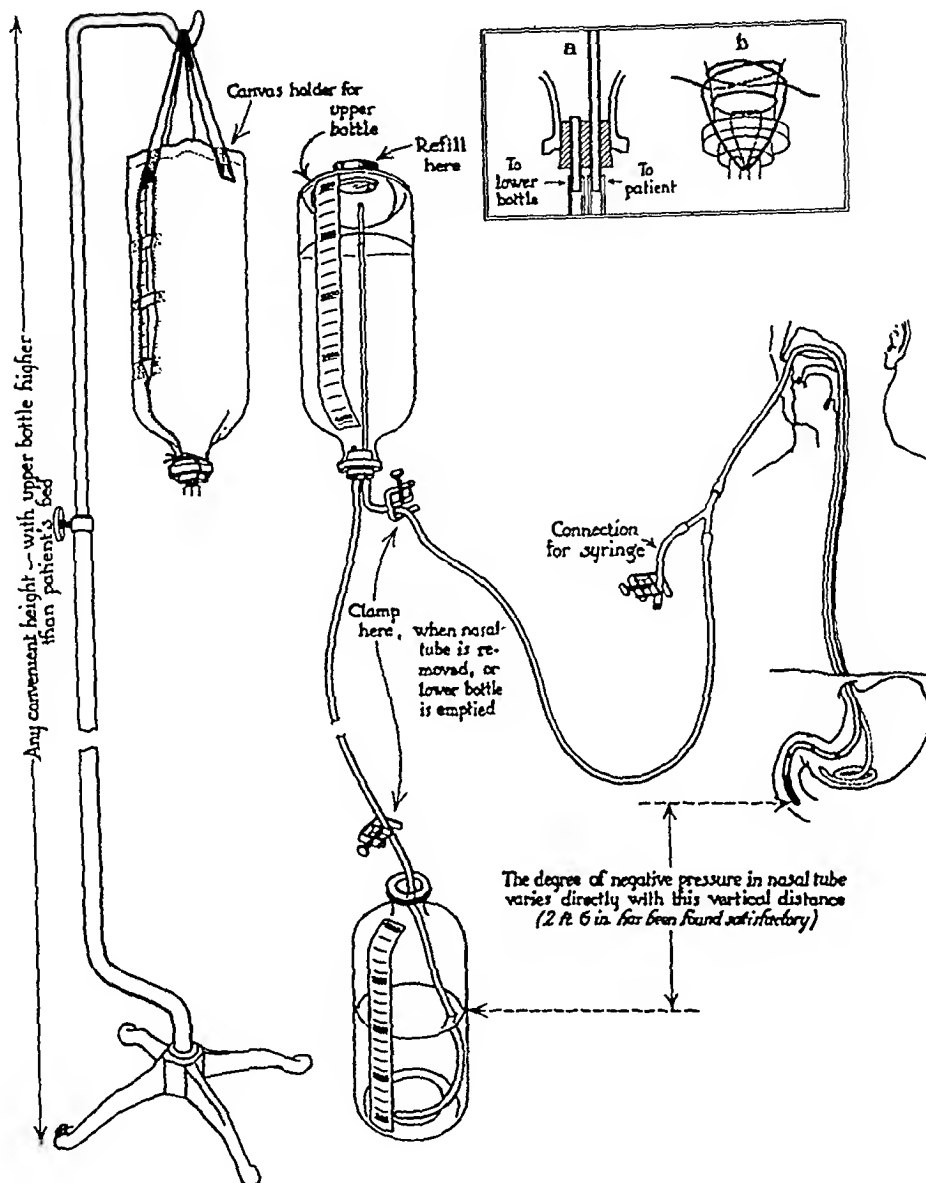

FIG. 1. Wangensteen apparatus for constant gastric or duodenal drainage.

Constant suction for decompression through a nasal catheter by the method of Wangensteen or related methods, accompanied by drinking 4 to 6 quarts of fluid per day, may be done when dilatation of the stomach is marked. (Fig. 1.) Hypochloremia must be especially guarded against if this treatment is continued over several days. In patients with retention who have had alkaline therapy, this continuous auto-lavage should be discontinued

in the gastric contents. If the gastric acidity is low or absent, dilute hydrochloric acid, four teaspoonfuls (16 c.c.) to a quart (1,000 c.c.) should be used in the gastric lavage.

Finney has advocated the "sterile" preparation of the stomach for operation by several days of sterile feeding and systematic lavage. All gastric cancers are more or less infected, especially when ulcerated and associated with achlorhydria;

it is practically impossible to sterilize such a stomach, particularly when pyloric obstruction is present. A valuable adjunct in this effort is the use of antiseptic throat gargles, nasal sprays and the removal of infected teeth with the institution of proper dental hygiene.

Preoperative Rest in Bed. Debilitated patients are kept in bed for one or two days prior to operation in order to help build up their resistance and facilitate the administration of fluids. The average patient, however, is not put to bed previous to the operative day because activity improves his morale, pulmonary ventilation and muscular and circulatory tone.

Preparation of the Diabetic Patient for Gastric Resection. Because of the usual malnourished state of diabetic patients with gastric cancer it is desirable to use a high caloric diet with adequate insulin to lower the blood sugar in a few days to a week. The usual diet is 150 Gm. of carbohydrate, 100 Gm. of fat and 75 Gm. of protein. The blood sugar is never lowered rapidly in patients with arteriosclerosis. The ideal preoperative blood sugar is 150 to 200 mg. per 100 c.c. The urine may contain a few tenths of 1 per cent of glucose but should contain no acetone bodies. In the presence of a high degree of obstruction with gastric retention other methods of giving food may be necessary and the diet will be more limited.

Fluids and extra glucose are administered the day before operation and on the day of operation no fluid is given by mouth for six hours before the patient is taken to the operating room. If acidosis and hyperglycemia are not controlled and insulin is necessary, intravenous glucose with insulin is administered.

At the Beekman Street Hospital, Terry has formulated a scheme for determining the amount of insulin needed by the color of the reduction in the urine: he gives 15 units if there is red reduction, 10 units if yellow reduction, and 5 units if green. This insulin is accompanied by orange juice or ginger ale by mouth. Immediately after the operation, Terry gives 20 units of insulin with 1,000 c.c. of 5 per cent glucose. One hour later he gives 5 to 10 units of insulin and discards the catheterized urine obtained at that time. Two hours later the urine is examined and insulin given according to his color formula stated above. Six hours later he gives intravenous 5 per cent glucose with insulin coverage. One hundred Gm. of carbohydrate are given each twenty-four hours postoperatively by some route.

Careful observation for any evidence of metabolic abnormality is imperative as it is better to err on the side of too little control rather than too much, as slight glycosuria is not so dangerous as insulin coma. The surgeon should be aware of the frequency of cardiac complications in diabetic patients, particularly during the postoperative state; such a complication may be easily confused with insulin reaction.

GENERAL CARE OF PATIENTS AFTER OPERATIONS FOR GASTRIC CANCER

The postoperative course depends largely on the nature of the operation, the skill and ease of its execution and the degree of care in the preoperative preparation of the patient.

Adhesive tape is never placed too tightly around the upper abdomen especially over the costal margin. Sufficient carbon dioxide (10 per cent) and oxygen mixture is given by inhalation for fifteen minutes hourly for the first eight to twelve hours, to cause hyperpnea. This ventilation of the lungs is continued by encouraging the patient to take twenty deep breaths four times daily or to continue the carbon dioxide and oxygen mixture three times daily for an additional three days.

Codeine ½ gr. is allowed every four hours for two or three doses, then every six hours for another four doses. Although morphine is allowed for young and for vigorous middle-aged patients, it is rigor-

ously withheld from most subjects over 50 years of age, at least during the first forty-eight hours. Such patients seldom die of shock or fatigue, whereas heavy morphinization is only too often followed by pneumonia. In the experience of the authors, it has seemed best to permit a certain degree of restlessness of the patient, providing suffering is not too intense.

Slight Trendelenburg position is desirable until the patient reacts from the anesthetic. This may be continued if there is a poor venous return from the extremities or if bronchiectasis is present. The position otherwise is changed in the afternoon following the operation to a quarter sitting position.

A warm enema (40°C.) may be given after twenty-four hours. When the patient wishes to cough, it is well for the nurse to support the rectus muscles. External heat from a hot water bottle or electric pad may relieve rectus spasm.

The recent routine postoperative use of drugs to stimulate intestinal musculature seems to be valuable in the prevention of gas pains, postoperative distention and paralytic ileus. In the selection of drugs, the discoveries of Puestow should be followed. He was able to study the influence of these agents on the visible peristaltic activity of the large and small intestine because of the herniation of the cecum and terminal ileum in a patient who had the ileocecal segment isolated from the remainder of the intestinal tract. In brief, morphine, eserine and prostigmin stimulated increased peristalsis in the small intestine while at the same time peristalsis of the large bowel was paralyzed. On the other hand, pituitrin and pitressin stimulated increased peristalsis in the large intestine while at the same time peristalsis of the small intestine was paralyzed. No drug was found to stimulate both small and large intestine. Therefore in the selection of drugs, the surgeon should choose the proper one to increase the tonus of either the small or large intestine depending on where the gas accumulates. When mor-

phine is used (in all but elderly patients) it is a most efficient stimulator of small intestinal motor activity, but should be followed first by enemas and later if necessary by pitressin to overcome the stasis in the large bowel. Potler and Mueller advocate the routine use of 1 c.c. (20 units) of pitressin to be given intramuscularly at the close of the operation and then repeated every four hours for six to ten doses. Where any peritonitis exists, the interval is two hours instead of four. If mechanical ileus develops it requires mechanical therapy, such as the use of the Abbott-Levine tube, ileostomy or operation to relieve the cause of the obstruction.

Diet. There are many postoperative diets in use throughout the United States today, and the majority are serviceable. The authors prefer, however, those postoperative diets in which small quantities of liquid food are given to the patient at frequent intervals immediately after the operation. There is no harm in early feeding, providing the surgeon is alert to the possibility of gastric dilatation. On the morning of the first three postoperative days a Levine tube is routinely inserted into the stomach, generally over a period of from thirty to sixty minutes, in order to remove any residual liquid. The following diet is one which was used years ago by Charles Peck of the Roosevelt Hospital in New York City. It is the standard diet employed at the Memorial Hospital for patients who have had gastric resection or gastroenterostomy.

As soon after operation as the patient is quiet, a 6 ounce 5 per cent glucose solution containing 40 gr. of sodium bromide, is administered as a retention enema.

Beginning four hours later, 6 ounces of tap water are given by rectum every four hours. This procedure is continued uninterruptedly for two days, if the fluid is retained.

Four hours after the operation, unless the patient is nauseated, 2 drams of hot water are given by mouth every two hours. This amount may be increased gradually until

as much as 1 ounce is given in the evening of the operative day.

First Day.   The day following operation, 1½ ounces of water and 1 ounce of chicken broth without salt or pepper, are allowed alternately every hour. At night, water and other fluids are given only when the patient is awake.

Second Day.   Add 1½ ounces of Martin's milk* every three hours and 1½ ounces of tea every four hours to the previously mentioned diet.

Third Day.   Allow water in small quantities as required. Give fluid food 3 ounces every hour. Add 2 ounces of strained gruel three times during the day.

Fourth Day.   Make the fluid food in quantities of 4 ounces at a time.

Fifth Day.   The fluid food is increased to 6 ounces every two hours. Junket and custard are added in small quantities three times during the day.

Sixth Day.   One coddled egg is added at luncheon. Milk of magnesia ½ ounce is administered at 9 A.M. and 6 P.M. after meals.

Seventh Day.   Toast, cereal and cream are added in small quantities.

Eighth, Ninth, Tenth and Eleventh Days. There is no increase of diet.

Twelfth Day.   A modified light selected diet is given; no crusts and no fruit, either raw or cooked, are allowed.

This diet is continued until the patient is discharged. The milk of magnesia is continued from the sixth day until the day of discharge, unless complications arise. The patients are not enjoined to follow a special diet after discharge from the hospital, as is the case after operations for gastric ulcer.

Postoperative Administration of Fluids. The amount and type of fluids required after operation should be such as to permit the ready continuance of normal body functions. The average daily secretion of digestive juices in man is 7,300 to 8,800 c.c.

* Martin's Milk is prepared as follows: Dissolve junket tablets in water and add to warm milk. Allow to cool and then strain. The whey is Martin's milk. Or it may be prepared by adding 1 dram of liquid rennet to 1 pint of warm milk.

According to Rowntree, this distribution is as follows: saliva—1,500 c.c.; gastric juice—2,000 to 3,000 c.c.; bile—300 to 500 c.c.; pancreatic juice—500 to 800 c.c.; intestinal juice—3,000 c.c.

The average intake of fluids in a normal individual with an average diet is approximately: drinking water—300 c.c.; liquid food—580 c.c; water content of solid foods—720 c.c.; water of oxidation from foods—300 c.c. The water of oxidation from fat is roughly twice as much as from the same amount of carbohydrate or protein. The measurement of postoperative fluid intake is accurate since the foods are all liquid for three or four days.

The loss of body fluids after operation is of great importance. In a normal person, the loss of water from the lungs is constantly about 300 c.c. daily; the loss through the kidneys varies with the intake, averaging 800 to 3,000 c.c.; the loss with feces is small, about 180 c.c.; but the skin eliminates a variable quantity since one of its functions is the dissipation of heat, and this loss of fluid amounts to one-third or one-half of the total extrarenal loss. The last factor is one which is often overlooked after operation.

Coller and Maddock have measured the actual fluid loss during operation. This must include loss by vomitus and hemorrhage in addition to the normal avenues of water elimination. The amount lost by vomitus during operation is not important, but the loss through hemorrhage is often greater than the surgeon believes, sometimes measuring 1,200 c.c. The patients observed who had operations on the stomach (one gastroenterostomy for carcinoma and one resection for carcinoma) lost 111 and 274 Gm. of blood respectively. The fluid elimination through the lungs and skin was at times as high as nine times that of the basal state, amounting to 1½ to 2 liters a day. A comparison of urinary output with the fluid intake did not give a correct estimate of water balance, because the urinary output averaged less than 10 per cent of the total fluid lost. Diarrhea

is another usually ignored source of fluid loss.

On the basis of his experiments Coller used 6 per cent of the total body weight in calculating the water requirement of the tissues in dehydrated patients. This estimation along with the consideration of the amount of fluid for vaporization, for abnormal losses and for a reasonable urine output gave him a rather exact indication of the amount of fluids needed to restore the dehydrated patient to a normal water balance. He considers a satisfactory urine output to be about 1,500 c.c. a day for sick patients as normal kidneys need to excrete about 500 c.c. of urine a day for normal function.

Maddock and Coller in a more recent article give the following table as the water requirement for a dehydrated patient weighing 60 Kg.

	C.c.
1. Water of vaporization	2,000
2. Water for urine	1,500
3. Abnormal loss during twenty-four hours	
4. Water to restore depleted fluids (6 per cent of 60 Kg.)	3,600
Total for twenty-four hours	7,100

Fantus made a statistical postoperative study of 2,000 patients at the Cook County Hospital, observing the quantity of urine, chlorides and glucose excreted. He concluded that patients should not be operated upon if they do not pass at least 1,500 c.c. of urine containing at least ½ of 1 per cent of chlorides the twenty-four hours preceding operation. If surgical treatment is necessary without these requirements the postoperative care will be more difficult. An excessively low or high salt elimination during the postoperative state is attended by a high mortality rate. Fantus also advised that very sick patients should not be given large quantities of fluid "unless grossly dehydrated."

Dextrose solutions are useful both to replenish the glycogen supply of the liver and to furnish nourishment. The action varies somewhat with the concentration and rate of administration. Five per cent solution of dextrose in water is an isotonic solution that is used chiefly to combat dehydration. More concentrated solutions of dextrose (10 to 50 per cent) are hypertonic and are eliminated fairly rapidly, thereby acting as a diuretic. If more than 0.4 Gm. of dextrose per pound of body weight per hour is administered, the carbohydrate will not be fully metabolized and is spilled into the urine, carrying water with it.

Salt solution acts chiefly in the blood to dilute the blood colloids and momentarily lower the osmotic pressure of the circulating fluid. An isotonic solution (0.85 per cent) after infusion or absorption into the blood stream is rapidly lost into the tissues, thereby restoring the water balance. Therefore it is not of much value in restoring and maintaining the volume of the circulating blood. It can on the contrary produce edema leading to convulsions, without an effective increase in the blood volume. Hypertonic salt solution (usually 2 per cent) introduced into the blood produces a transient return of fluid from the tissues into the blood stream but the balance is rapidly restored to normal because of the diffusibility of salt. Salt is not metabolized and a high salt content in the plasma interferes with the excretion of fluid through the kidneys, causing retention with demonstrable edema.

Ringer's solution contains in 1 liter of distilled water, sodium chloride 7 Gm., calcium chloride 0.25 Gm., potassium chloride 0.30 Gm. It is a buffer solution which has been preferred for intravenous infusions and its use will be discussed in that connection.

Hartmann's solution has the same constituents as Ringer's solution with the addition of sodium bicarbonate and dextrose. This buffer solution serves to restore the water balance, restore chlorides, replenish sodium, potassium and calcium ions in the proper proportions as well as supplying the sodium bicarbonate. It is especially indicated for pyloric obstruction, intestinal obstruction, gastric and intestinal fistulae.

METHODS OF ADMINISTRATION OF FLUIDS

Oral Method. Although unquestionably the best method of giving fluids, the intake by mouth must be restricted during the period of gastric wound healing. Small quantities of water and clear nourishing liquids are given at frequent intervals immediately after the operation. (See diet—page 440.) The suture line at this time is less likely to become disrupted than it is on the third and fourth postoperative day.

Intravenous Infusion. The technique of continuous intravenous drip for postoperative administration of liquids was described by Matas in 1924. He cited its use during operation with the primary massive introduction of isotonic solution followed by a continued drip of 5 per cent dextrose in water averaging forty to sixty drops per minute. Horsley in 1927 advocated the use of this method for administering 10 per cent glucose in Ringer's solution pre- and postoperatively in amounts ranging from 1,000 to 1,500 c.c., giving it at the rate of 150 to 250 c.c. per hour. In 1931 Horsley modified the type of cannula used for continuous intravenous infusion and changed to 5 per cent glucose in Ringer's solution for the usual patient and to 5 per cent glucose in distilled water for patients with kidney disease and edema. A transfusion of citrated blood may be given through the same cannula, while continuous intravenous administration of fluids is in progress. The physician should be alert for the manifestation of "water sickness" such as puffiness of the lower eyelids, unusual lacrimation and vomiting of clear or bile-stained fluid.

If the hospital is not equipped with a special unit to prepare solutions for intravenous use, then commercial solutions should be used. The solutions should be prepared properly and accurately, using pure chemicals and storing them in an aseptic vacuum bottle which should be dated, and not used when more than a week old. The exact measurement of correct temperature of the fluid is not important, if the solution is given slowly enough, as for example, 4 to 6 c.c. a minute. There may be some danger of embolism and thrombosis following a long intravenous administration of fluids, but this complication has not occurred in the writers' experience.

Recent work by Coller and Maddock, showing the effect of different solutions given intravenously to sick surgical patients for a number of days, will be briefly cited.

1. A group of patients were given 5 per cent glucose in normal salt solution, and all gained weight and retained water. If this was discontinued for oral administrations, or changed to 5 per cent glucose in water, the retained water was lost in all cases.

2. Five per cent glucose in Ringer's solution was given, and six of the seven patients had fluid retention. This also was lost when discontinued or the solution changed.

3. Five per cent glucose in water, given to a third group of sick surgical patients, caused no retention of water. Some patients lost weight due to utilization of body tissues for energy. It was concluded that 5 per cent glucose in distilled water provides for a normal water exchange, and the only reason that gross water retention was not seen more frequently with the indiscriminate use of saline solution was that it is generally not given for more than two or three days.

Coller and Maddock then studied the reasons for giving parenteral fluid in 100 surgical cases. In 20 per cent there was vomiting, and salt was therefore needed. The amount needed was determined in one of two ways: the vomitus was measured in some cases and an equal amount of Ringer's solution given. They preferred Ringer's solution for patients needing sodium chloride. In other patients, the blood chlorides and carbon dioxide combining power were determined. If these values were low, Ringer's solution was given in quantities of 1½ to 2 liters a day. The Ringer's

solution was alternated liter for liter with 5 per cent glucose in water. The duration of this treatment was determined by blood chemical studies every forty-eight hours. The other 80 per cent of patients were given parenteral fluids because they were unable to take sufficient fluids by mouth. In these cases, it was considered that solutions containing sodium chloride were unnecessary and dangerous.

Hypodermoclysis. Hypodermoclysis as a method of fluid administration is still used, but not to so great an extent as formerly, because of the more common employment of intravenous infusion over a continuous period. When this method is employed, it should be preceded by novocaine infiltration of the skin at the site of the introduction of the needle. In addition, 10 to 20 c.c. of 1 per cent novocaine solution may be added to the body of the fluid, in order to maintain continuous analgesic effect. Woodyatt's method, advocated several years ago, of using multiple small needles causes very little discomfort, and is deserving of more common usage. The subcutaneous tissues of the thigh are preferred to the subpectoral region because there is less embarrassment to respiration.

Proctoclysis. The rectal method of administration of fluids and glucose has generally fallen into disrepute and disuse. McNealy and Williams, in 1924, experimented with dogs and showed that there was no appreciable absorption of 5 per cent aqueous solution of glucose from the colon, although there was some absorption from the ileum. Pressman found in humans a fall in peripheral blood sugar after giving glucose per rectum. The findings were confirmed by de Takats and Scott and Zweighaft. Perusse found in dogs that a 1 per cent glucose solution was the ideal fluid, given rectally, for restoring water balance. It would seem that the total amount of dextrose absorbed from the colon under the best conditions is too small for any considerable immediate therapeutic effect. The rectal administration of fluid is indeed a very inefficient substitute for the oral route. Tap water alone may be given either by the Murphy drip or by the introduction of 4 to 6 ounces at a time, every three or four hours, as tolerated by the patient. The rectum and colon become intolerant of even the fluid, after twenty-four to thirty-six hours. The purpose of proctoclysis therefore is solely for the absorption of water, and not as a nutrient enema.

HEALING OF THE WOUND

A surgical wound is termed by Bigelow a self-limited disease. The strength of sutured wounds in skin, muscle, fascia, and gastric mucosa has been studied by Howes and his co-workers. During the first four days there is very little healing in all tissues, but during the first two days the wound is stronger than on the third or fourth day because of the strength of the suture material. Early healing is only an agglutination of tissues. The tensile strength of the agglutinated wound is not greater than that of the finest catgut. The weakness of the wound on the third and fourth days is due to autolysis and pressure necrosis around the sutures and varies with the amount of this reaction. Therefore, the least amount of trauma and of suture material necessary to approximate the tissues is most desirable. After the fourth to sixth day proliferation of the fibroblasts and endothelium strengthens the wound at first rather rapidly and then more slowly to a maximum strength in ten to fourteen days. This maximum strength is reached slightly later in fascia than in skin and muscle. Infection is the principal factor that retards the healing process.

The mode of healing of gastrointestinal anastomoses in dogs has been carefully studied by Flint. Healing by first intention may be complete in five to fourteen days, but it is not the usual occurrence. More often there is considerable exudation, destruction of tissues, demarcation of necrotic tissues and organization of exudate with regeneration of the mucous membrane, requiring the full fourteen days for

complete wound healing. The mucosa of the stomach is flattened and grows up and around or through the exudate, covering it before it grows down to form glands. As the cells of the mucosa grow older some with acidophilic reaction appear, increase in size and form young parietal cells while the other cells become chief cells. The muscularis mucosa may regenerate from itself, or cells may grow up from the inner circular muscle layer to form it. If this does not occur, the glands will push down into the submucosa. The muscular layers regenerate from the torn ends chiefly from the outer oblique layer, but considerable of the union between the muscles is accomplished by fibroblasts rather than myoblasts.

POSTOPERATIVE PULMONARY COMPLICATIONS

The pulmonary complications of gastric surgery occur more frequently in males than in females. Keen's statistics showed 9.1 per cent such complications in males as compared with 4.5 per cent in females. Eliason and McLaughlin found that 70 per cent of the patients in their series in whom these complications occurred were males.

It is generally believed that the type of anesthesia does not influence the incidence of pulmonary complications to any great degree; some reports indicate practically as high an incidence after spinal and local anesthesia as after the administration of general anesthesia. Rovenstine has found that the most important predisposing factor has to do with the plane of anesthesia. The deepest plane of anesthesia continued for an appreciable length of time doubles the incidence that is found in patients when analgesia as the first plane of anesthesia only is necessary. The duration of the anesthesia is also important, according to Rovenstine; when the time is more than ninety minutes the incidence becomes higher than the average incidence, and increases rapidly with every added half-hour. The authors' experience is contrary to general opinion, in that the frequency of pulmonary complications has been less

than 6 per cent with the routine adoption of spinal anesthesia for gastric resection, or palliative operations for gastric cancer.

There is also a definite seasonal variation in the frequency of postoperative pulmonary diseases as there is in the incidence of pneumonia in the general population, the greatest incidence, as a rule, being in February, March and November. The occurrence of intercurrent illnesses such as oral sepsis, cough, pharyngitis, emphysema and allergic conditions, render the patient more susceptible to complications in the respiratory tract.

The most important influencing factor, however, is the type of operative procedure. Because of the frequent occurrence of pulmonary diseases after operation on the stomach and duodenum, a rather complete discussion of this subject seems desirable. The mortality of these complications varies from 25 per cent to 50 per cent of the patients in whom they occur.

There has never been adopted a suitable appellation of these postoperative pulmonary complications, as there are many different terms employed for the same ailment. Matas refers to them as "postoperative pneumopathies" after the French school; Whipple considers the common complication an aspiration pneumonitis; Cutler considers it as an embolic infarction; whereas Lee, Corrylos, Jackson refer to it as "atelectatic pneumonia." However, the definite clinical course and radiographic findings suggest that these complications may be classified as entities.

Embolism of the pulmonary artery is a late postoperative complication, usually with symptoms which are characteristic: shock, pain and hemoptysis. The signs, course and prognosis will depend upon the size of the artery which is occluded. If it is of considerable size, the lung which becomes consolidated may have a wedge-shaped shadow on the Roentgen film. Small pulmonary vessels are probably involved more frequently than is generally recognized.

Postoperative lobar pneumonia occurs infrequently. It is caused by the pneumo-

coccus which should be typed from the sputum, after which the pneumonia is treated just as the usual medical case of this disease.

Aspiration pneumonia and hypostatic pneumonia occurring after operation are more difficult to diagnose. There may be physical and radiographic signs of consolidation and a typical clinical course; or the signs may be vague and the clinical course rapid. Many of the pulmonary complications do not fit any of the other types named and probably belong in this heterogeneous group.

Massive collapse of the lung occurs in about 1 per cent of operations for gastric cancer. It may be partial or complete. The clinical symptoms and signs with characteristic x-ray findings make the diagnosis less difficult. There is considerable evidence that atelectasis occurs more frequently in the lung postoperatively than does a real pneumonic process. It has been shown that chest expansion is reduced 75 per cent after abdominal operations; supero-inferior diameter of the thorax is greatly diminished due to marked elevation of the diaphragm. In 75 per cent of the patients with no clinical evidence of pulmonary complications, Muller, Pendergrass and Overholt found an increased density of the trunk shadows producing a triangular area of density in the lower lobe. The vital capacity following abdominal operations has shown a reduction of 64 per cent of the preoperative or normal values in a series of 218 patients. Churchill and McNeall, Head and Powers have made similar observations.

In addition to the factor of pulmonary hypoventilation in the production of pulmonary atelectasis, the factor of bronchial obstruction is also important. Lee, Jackson and Coryllos believe that postoperative pneumonias develop in atelectatic portions of the lung produced by bronchial obstruction. In allergic patients, due to the physical shock of the surgical procedure or the protein shock of the extravasated blood or bacteria, excessive bronchial secretion added to the edema of the mucous membrane or bronchial spasm results in obstruction. This obstruction is not completely relieved by coughing, since the cough reflex is usually abolished or diminished by narcosis. Therefore, it continues and the secretion becomes more viscid. There is said to be a ball-valve action, allowing the escape of air with expiration, but no entrance with inhalation or complete plugging with absorption of the confined gases. The rapidity with which atelectasis becomes complete depends on the mechanism and the percentage of oxygen in the confined air, as it is absorbed more rapidly than nitrogen.

In the treatment of pulmonary complications, several prophylactic measures are adopted. The first, of course, is to avoid the predisposing factors. The routine postoperative use of a mixture of 7 per cent to 10 per cent carbon dioxide with 90 per cent to 93 per cent oxygen, has been generally accepted and utilized. It is our practice to administer this mixture of gases every two hours during the first thirty-six hours following the operation, except when the patient is sleeping and even more frequently during the first eight to ten hours. The gas is given in sufficient quantities to cause hyperpnea. It is also advisable to institute deep breathing exercises as soon as the patient can coöperate. Other prophylactic measures are the avoidance of excessive narcosis, the avoidance of tight-fitting bandages on the upper abdomen, frequent change of posture of the patient and the prompt relief of abdominal distension. When such complications do occur, oxygen inhalations are doubly indicated. The great value of bronchoscopy and direct bronchial aspiration through this instrument is so generally recognized that it need not be stressed.

SHOCK

This term is quite generally believed to be unsatisfactory, but a suitable substitute has not been found for the clinical syndrome that is familiar to all, the confusion being due: first, to the obscurity of the cause of shock, and second, to the usual

failure to differentiate between primary and secondary shock (termed psychogenic and neurogenic by Phemister). The typical picture of this condition is a rapid and thready pulse, rapid and shallow breathing, loss of motor reflexes and a lowered basal metabolism, giving a subnormal temperature.

In primary shock or collapse, Goltz found that the essential change was vaso-dilatation, due to reflex cardiac and vasomotor paralysis. The lowered blood pressure may persist from a few minutes to an hour or more, after which recovery may take place spontaneously, or following the administration of drugs. There is little or no change in blood volume. If recovery does not take place, the patient passes into secondary shock.

In secondary shock, the profound circulatory depression is not due to heart failure, nor is it due to vasomotor paralysis, since it has been shown experimentally that the vasomotor reflexes are normal during shock, and there is often no cardiac disease. The blood shows a relative increase in hemoglobin content and higher viscosity, with diminished volume in the blood vessels, and diminished alkaline reserve. A clinically analogous condition is found in histamine poisoning, which is known to paralyze the capillaries in a state of dilatation while constricting the arterioles. The dilated capillaries are more permeable than normal and fluid containing serum protein is lost into the tissues, resulting in diminution of blood volume with increased viscosity. A lowered alkaline reserve or slight acidosis results from impaired oxidation in the tissues. It is not great enough in amount, as a rule, to produce detectable edema. The site of the capillary dilatation is not known; the skin is very pale, definitely excluding the peripheral capillaries, and the splanchnic vessels are not found engorged during operative procedures when shock supervenes. The best explanation at present seems to be that the concentration is in the skeletal muscles or else that the loss is only in "effective blood volume."

Although this mechanism and these findings are generally accepted for the condition termed shock, the etiologic factor or factors are much disputed by experimental workers and probably there are various forms with different specific causes. The acceptance of histamine or a toxin as the possible cause in most cases is not general, although Cannon has warned against discarding this theory altogether. There are various contributory factors that may be partly responsible for the production and maintenance of shock. Among these may be mentioned starvation, dehydration, exposure to cold, unusually prolonged anesthesia, excessive loss of blood, infection and trauma.

The treatment is first and foremost to avoid the predisposing causes. The curative treatment cannot be specific because the cause is not known. Therefore, one may attempt first to replace the blood volume and, second, to produce capillary constriction. Blood is the best solution to replace the volume of circulating fluid as it replaces the lost serum protein and is less rapidly diffused from the dilated capillaries. Other colloid containing fluids such as gum acacia and gelatin solutions have been considered the next choice. Acacia is now available in a refined form and in convenient containers for intravenous use as prepared by several of the larger drug houses. It is usually administered in amounts of 300 to 600 c.c. of a 6 per cent solution. It cannot be added to citrated blood as clotting would occur. Erlanger and Gasser have shown that acacia solution reduces the plasma loss, but it has been reported that it may cause damage to the liver.

Saline solutions are of little value, since permeation of these fluids through the capillaries will lead to further interference with oxidation in the tissues. Sodium bicarbonate solutions have been used because of the associated acidosis in shock; but they are dangerous because an uncom-

pensated alkalosis may result. Ten per cent glucose solution, even though the effect of its hypertonicity is temporary, is probably the best solution to replace blood volume if acacia or gelatin solutions are not available. Frazier has reported beneficial results from the administration of 10 per cent ethyl alcohol in 10 per cent dextrose, given very slowly intravenously. He states that 2 liters may be given in twenty-four hours, and that the patient experiences a feeling of warmth and well-being. The application of external heat and lowering the head are general measures that hardly require mention.

Drugs that are known to act as vaso-constrictors, such as adrenalin or pitressin have no value in doses which can be used safely. A temporary rise in blood pressure which may be produced may be followed by a worse depression. Adrenal cortex or cortin has been reported by Andrus and Heuer to give encouraging results. Caffeine has very little value as a stimulant in this condition.

If anesthesia has played a rôle in producing the state of shock and respiration ceases, it must be remembered that the normal stimulus of respiration is not the lack of oxygen, but the presence of carbon dioxide. With rapid respiration, the carbon dioxide is eliminated from the blood stream, and acapnia as described by Yandell Henderson, will be present.

OTHER COMPLICATIONS

Hiccough. Hiccough or singultus is due to a clonic spasm of the diaphragm. When it occurs postoperatively and is persistent, it is not only a great discomfort, but an alarming symptom. It occurs most frequently when the abdominal viscera and their peritoneal coverings have been disturbed or irritated. It may appear as one of the first signs of acute dilatation of the stomach. The afferent impulses are carried by the phrenic nerve and the sympathetic trunks. It seems to occur more frequently in females, often over 45 years of age.

The treatment is sometimes difficult. Gastric lavage and continuous gastric drainage may be beneficial. Morphine in large doses will usually aid in causing sleep, with abatement of the hiccoughs. If it is severe, no foods or fluids are allowed by mouth. If these methods fail, and the symptoms become intractible, either injection of the phrenic nerve with alcohol or exposure of the nerve and slight squeezing with the hemostat, may be used as a last resort to maintain life. A test of the effectiveness of this procedure is to inject the phrenic nerve with novocaine to see if there is an immediate but temporary abolition of the hiccoughs.

Parotitis. Parotitis is a distressing and often fatal complication following operation for gastric cancer. After it appears, the progression from an acute inflammation of the acini of the gland to suppuration occurs very rapidly, since the debilitated state of the patient offers no barrier to the infective agent.

Three predisposing factors can be considered and eliminated in part. Parotitis occurs more frequently in patients who are dehydrated; therefore, this condition should not be permitted to develop during the postoperative state. The incidence of this complication increases with patients who are not ingesting liquids by mouth, and consequently there is a tendency for stagnation of salivary secretion. A certain degree of stomatitis often develops in patients who have been on a long continued liquid diet.

As a prophylactic measure, salivary secretion may be induced by giving the patient lemon peel to chew three or four times daily. When the parotitis develops, the early institution of radiation therapy, either with radium or Roentgen rays, may be of definite value in aborting the infection. Repeated suberythema doses are administered. Large doses of potassium iodide are indicated. In some instances, it has been found worth while to cannulize Stenson's duct and irrigate with dilute acriflavine or Dakin's solution.

Acute Dilatation of the Stomach. This complication may occur after any operation on the stomach, usually during the first forty-eight hours. The occurrence of hiccoughs, an unusually rapid pulse, upper abdominal distention, regurgitation of fluid, loss of desire for water, and peculiar facies may indicate the presence of this condition. It is most important that it be recognized early. The stomach will usually contain an amount of fluid which is far in excess of the fluid intake; it practically always contains bile and little or no hydrochloric acid. If this fluid is removed, and the stomach lavaged until the fluid return is clear immediately after recognition of the complication, there is rapid recovery. No fluids should be given by mouth. Morphine for the induction of sleep in helpful. Change of posture to relieve the pressure on the superior mesenteric artery has been advised. A Levine tube introduced through one nostril is left in place to provide continuous drainage and decompression of the stomach for at least twelve hours. Prostigmin or eserine is given hypodermically every three hours for three or four doses.

Postoperative Gastric Hemorrhage. This complication occurs early, usually within the first forty-eight hours after gastric resection. It is manifested by an appreciable amount of bright red blood in the vomitus. After morphine has been given to assure rest and allay nervous excitement, the organ must be evacuated of its contents and put at rest also. Repeated lavage with water at 120°F. has been advised by Young and Fowler as early as twelve hours after operation. Finsterer and Cunha first attempt to control such bleeding by lavage with ice water, and if this is unsuccessful, they introduce 100 c.c. of 1:1,000 silver nitrate solution into the stomach; this procedure may be repeated only once. The intravenous administration of calcium and the subcutaneous injection of horse serum has been advocated, but are of questionable value. The parenteral administration of fluid, or blood transfusion, permits the replacement of the fluid loss in such complications, but these measures are not carried out, as a rule, until the bleeding has definitely stopped or unless the loss from hemorrhage is alarming.

Postoperative Peritonitis. In the general experience of the abdominal surgeon this complication is the cause of one-fourth to one-tenth of the fatalities after gastrointestinal surgery. It may be due to unavoidable contamination from badly infected gastric contents; on the other hand, it may be due to gross leakage of gastric contents from defective anastomoses, perforation elsewhere in the gastrointestinal tract, gangrene and necrosis from impaired blood supply, or from a suppurative focus in the wound. The best treatment is prevention by careful and prolonged preoperative preparation of the patient, and by meticulous care in the technique of resection and anastomosis. The treatment of peritonitis may be found in any of the general surgical textbooks. In general it depends upon the cause of the peritonitis and the stage and extent of this complication.

Postoperative Diarrhea. Postoperative diarrhea is a distressing complication and, if uncontrollable, may be fatal. The etiology is unknown. It is generally believed to be due to proctitis and colitis. In Beer's experience, the cause may be too large a gastrointestinal stoma, or a localized peritonitis. Anschütz studied this complication in 500 patients who had gastric operations; he found that it occurred early in some cases, but usually on the seventh to the tenth day. The hazard of a fatal outcome is increased if the diarrhea follows the performance of a gastroenterostomy in the absence of free hydrochloric acid in the stomach. Inflammation of the jejunum characterized by irritation of the mucosa may follow too rapid emptying of the stomach.

Bierende studied seven cases with this complication, following various operations, three of which were for ulcer and one for carcinoma of the esophagus. The pathologic changes were chiefly in the rectum and upper colon. At first these changes consisted of small, confluent, hyperemic plaques on

the rugae. Later, desquamation and coagulation necrosis occurred, the fibrin in the necrotic regions giving a honeycombed appearance to the surface. Goldschmidt and Muelleder found postoperative colitis or enteritis occurring in 29.3 per cent of their patients who had gastric operations.

The treatment of this postoperative diarrhea, in its mild forms, is the institution of a simple non-irritating diet and astringent therapy. The administration of large quantities of dilute hydrochloric acid by mouth, together with large doses of opiates, may be necessary. It is possible that the incidence of this complication may be lowered by the avoidance of prolonged preoperative catharsis.

Thrombophlebitis. This complication is not more frequent after gastric and duodenal operations than after other abdominal operative procedures. Nygaard has advised the following measures for prophylaxis; treat varicosities of the legs preoperatively or bandage the legs, avoid fluid enemas, maintain the body heat, encourage exercise of the extremities, elevate the leg on soft pillows, administer desiccated thyroid extract 2 gr. three times daily as soon as tolerated by mouth and continue until the patient is able to get out of bed.

Internal Hernia. This complication is a rare one following gastric resection or anastomosis. Buchanan, however, reported a case in which herniation of the jejunum between the ligament of Treitz and a gastrojejunal anastomosis had occurred. Of the three others reported in medical literature, all had occurred within three weeks after gastroenterostomy. The symptoms are pain, collapse, vomiting of large quantities of bile and other symptoms of intestinal obstruction. The treatment is operative, as soon as the indications are apparent. Preventive measures consist of obliterating the natural fissures by suturing the proximal loop to the lower part of the mesocolon.

Postoperative External Fistulae. Duodenal fistula is a very distressing complication of gastrointestinal surgery because of the digestive activity of the secretions on the abdominal wall. There are two principles of treatment: first the protection of the skin of the abdomen, and second, the neutralization or inactivation of the intestinal and pancreatic juices. The latter procedure is more important; it is accomplished by inserting one or more Dakin tubes into the interior of the wound, even into the duodenum, if possible. Through this tube is instilled by the Murphy drip principle a mixture of tenth normal hydrochloric acid containing Witte's peptone in solution. This neutralization of the alkalinity of the intestinal and pancreatic juices inactivates the strong proteolytic and lipolytic enzymes, so that they are no longer capable of digesting the living tissues of the abdominal wall. The peptone seems to serve as a fixative for the proteolytic enzymes, or a more ready source for their action than are the living tissues. The protection of the abdominal wall is of secondary importance; it may be accomplished by the liberal use of Kaolin or Fuller's earth, or finely powdered copper, or aluminum bronze paint; or by the application of a glue made by evaporating compound tincture of benzoin to the proper consistency. A fistula so treated may heal spontaneously and not require secondary closure. Postoperative gastric fistulae are less troublesome and will usually close spontaneously, providing the stoma previously made is sufficiently large to care for the major portion of the fluid intake.

REFERENCES

1. Allison, P. R. Continuous intravenous infusion. *Brit. J. Surg.*, 24: 137, 1936.
2. Andrews, E. Surgical shock. *Northwest Med.*, 34: 122, 1935.
3. Anschütz, M. Ueber die Damstorungen nach Magenoperationen. *Therap. de Gegenw.*, 45: 509, 1904.
4. Austin, J. H. Hydrogen ion concentration of the blood in health and disease. *Medicine*, 4: 275, 1925.
5. Balfour, D. C. Fundamental principles in surgery of the stomach and duodenum; report of four hundred cases. *Surg., Gynec. & Obst.*, 42: 167, 1926.
6. Balfour, D. C. Value of cooperation between internist and surgeon in the management of complicated gastric conditions. *J. A. M. A.*, 84: 876, 1925.

7. BALFOUR, D. C. Management of lesions of the stomach and duodenum complicated by hemorrhage. *J. A. M. A.*, 89: 1656, 1927.

8. BALFOUR, D. C., and HARGIS, E. H. Cancer of the stomach. *Am. J. M. Sc.*, 173: 773, 1927.

9. BEER, E. The significance of diarrhea following abdominal operations. *Ann. Surg.*, 77: 524, 1923.

10. BIERENDE, F. Ueber postoperative proctitis u. colitis. *Mitt. a.d. Grenzgeb. d. Med. u. Chir.*, 32: 85, 1920.

11. BLALOCK, A. Shock and hemorrhage. *Bull. New York Acad. Med.*, 12: 610, 1936.

12. BLOCK, F. B. Acute intestinal obstruction. *S. Clin. North America*, 12: 1473, 1932.

13. BROWN, G. E., EUSTERMANN, G. B., HARTMAN, H. R., and ROWNTREE, L. G. Toxic nephritis in pyloric and duodenal obstruction. *Arch. Int. Med.*, 32: 425, 1923.

14. BROWN, H. H. Value of feeding during operation as prevention of surgical shock. *Brit. M. J.*, 1: 510, 1917.

15. BUCHANAN, E. P. Internal hernia following posterior gastroenterostomy. *Ann. Surg.*, 96: 359, 1932.

16. CABOT, H. Water (Editorial). *Surg., Gynec. & Obst.*, 63: 681, 1936.

17. CABOT, H. Postoperative lung complications. *Surg., Gynec. & Obst.*, 52: 120, 1931.

18. CHRISTOPHER, F., and SHAFFER, J. M. Postoperative atelectasis. *Am. J. Surg.*, 32: 197, 1936.

19. CLUTE, H. M. The selection and management of patients for gastric surgery. *New England J. Med.*, 200: 580, 1929.

20. COBURN, R. C. Minimizing early postoperative pneumonia. *J. A. M. A.*, 100: 1392, 1933.

21. COLLER, F. A. Water balance in surgical patients. *Nebraska State M. J.*, 20: 365, 1935.

22. COLLER, F. A., and MADDOCK, W. G. Dehydration attendant on surgical operations. *J. A. M. A.*, 99: 875, 1932.

23. COLLER, F. A. Dehydration (Editorial). *Surg., Gynec. & Obst.*, 63: 249, 1936.

24. Co TUI, McCLOSKEY, K. L., SCHRIFT, M. H. and YATES, A. L. Filtration studies on reactive infusion fluids. *Proc. Soc. Exper. Biol. & Med.*, 35: 297, 1936.

25. Co TUI, McCLOSKEY, K. L., SCHRIFT, M. H. and YATES, A. L. The new method of preparing infusion fluids. *J. A. M. A.*, in press.

26. COWGILL, G. R. The Vitamin B Requirement of Man. Chapt. xv. New Haven, Conn. 1934. Yale Univ. Press.

27. CUTTING, R. A. Preoperative and postoperative care of gastric cases. *Am. J. Surg.*, 14: 573, 1931.

28. CUTTING, R. A. Absorption of dextrose and water by the small intestine and the colon. An experimental study. *Arch. Surg.*, 29: 643, 1934.

29. CUTTING, R. A. Principles of Preoperative and Postoperative Treatment. New York, 1935. Paul B. Hoeber.

30. DAVIDSON, J. R. An attempt to inhibit development of tar carcinoma in mice. *Canad. M. A. J.*, 32: 364, 1935.

31. DAVIS, H. A., and DRAGSTEDT, L. R. Relative significance of electrolyte concentration and

32. DOAN, C. A. Clinical implications of experimental hematology. *Medicine*, 10: 323, 1931.

33. EBELING, W. W. Absorption of dextrose from the colon. *Arch. Surg.*, 26: 134, 1933.

34. ELIASON, E. L., and McLAUGHLIN, C. Postoperative pulmonary complications. *Surg., Gynec. & Obst.*, 55: 716, 1932.

35. EUSTERMAN, G. B. Treatment following operation for ulcer of the duodenum and stomach. *Surg., Gynec. & Obst.*, 42: 161, 1926.

36. EUSTERMAN, G. B., and BALFOUR, D. C. Stomach and Duodenum. Philadelphia, 1935. W. B. Saunders.

37. FALK, H. C. The common causes of reaction following the use of intravenous solutions and their prevention. *New York State J. Med.*, 35: 480, 1935.

38. FANTUS, B. Fluid postoperatively a statistical study. *J. A. M. A.*, 107: 14, 1936.

39. FINNEY, J. M. Personal communication.

40. FINSTERER, H. and CUNHA, F. Surgical treatment of duodenal ulcer. *Surg., Gynec. & Obst.*, 52: 1099, 1931.

41. FLINT, J. M. The healing of gastro-intestinal anastomoses. *Ann. Surg.*, 65: 202, 1917.

42. GAMBLE, J. L., and ROSS, S. G. The factors in the dehydration following pyloric obstruction. *J. Clin. Investigation*, 1: 403, 1924–1925.

43. GOLDSCHMIDT, W., and MUELLEDER, A. Ueber postoperative Darmstorungen mit besonderer Berucksichtigung der Colitis. *Mitt. a.d. Grenzgeb.*, 32: 567, 1920.

44. GRAY, H. E. Postoperative pulmonary complications and postoperative use of the Trendelenburg position. *Minnesota Med.*, 18: 273, 1935.

45. HADEN, R. L., and ORR, T. G. Chemical changes in the blood of the dog after pyloric obstruction. *J. Exper. Med.*, 37: 377, 1923.

46. HARRIS, S. The management of ulcer patients before and after operations. Read before Am. Coll. Surg. Omaha, Neb., Feb. 4, 1930.

47. HARTMANN, A. F., and SMYTH, F. S. Chemical changes in the body occurring as a result of vomiting. *Am. J. Dis. Child.*, 32: 1, 1926.

48. HARTMANN, A. F. Acidosis, alkalosis and dehydration. *Colorado Med.*, 26: 373, 1929.

49. HARTMAN, H. R. Medical care of peptic ulcer. *Calif. & West. Med.*, 32: 5, 1930.

50. HARVEY, S. C., and HOWES, E. L. The treatment of wounds. *Yale Biol. & M. J.*, 1: 79, 1928.

51. HASTINGS, A. B., MURRAY, C. D., and MURRAY, H. A., JR. Certain chemical changes in the blood after pyloric obstruction in dogs. *J. Biol. Chem.*, 46: 223, 1921.

52. HENDERSON, Y., HAGGARD, H. W., and COBURN, R. C. The therapeutic use of carbon dioxide after anesthesia and operation. *J. A. M. A.*, 74: 783, 1920.

53. HENDERSON, Y. Atelectasis, massive collapse and related postoperative conditions. *Bull. New York Acad. Med.*, 11: 639, 1935.

54. HORSLEY, J. S. Partial gastrectomy. *Surg., Gynec. & Obst.*, 44: 214, 1927.

55. HORSLEY, J. S. Some underlying principles of intestinal surgery. *Ann. Surg.*, 87: 387, 1928.

56. Horsley, J. S. Modern methods of preoperative and postoperative treatment. *J. Indiana State M. A.*, 22: 424, 1929.

57. Horsley, J. S., and Horsley, G. W. Continuous intravenous injection of dextrose in Ringer's solution. *Arch. Surg.*, 22: 86, 1931.

58. Horsley, J. S. Further observations on continuous intravenous injection of dextrose in Ringer's solution. *Virginia Med. Monthly*, 61: 84, 1934.

59. Howes, E. L., Sooy, J. W., and Harvey, S. C. The healing of wounds as determined by their tensile strength. *J. A. M. A.*, 92: 42, 1929.

60. Howes, E. L., and Harvey, S. C. The strength of the healing wound in relation to the holding strength of the catgut suture. *New England J. Med.*, 200: 1285, 1929.

61. Howes, E. L., and Harvey, S. C. Recent studies in wound healing. *Yale J. Biol. & Med.*, 2: 285, 1930.

62. Kellogg, F., Mettier, S. R., and Purviance, K. Studies on hypochromic anemia in dogs. 1. The evaluation of a standard bread diet and of a meat diet on formation of hemoglobin before and after gastrectomy. *J. Clin. Investigation*, 15: 241, 1936.

63. Kendall, H. Atropine and its derivatives in gastroenterology. Novatropine. *Clin. Med. & Surg.*, 41: 205, 1934.

64. King, D. S. Postoperative pulmonary complications. 1. Statistical study based on two years personal observation. *Surg., Gynec. & Obst.*, 56: 43, 1933.

65. King, D. S. Postoperative pulmonary complications. 2. Carbon dioxide as a preventive in a controlled series. *J. A. M. A.*, 100: 21, 1933.

66. King, D. S. Postoperative pulmonary complications: the part played by anesthesia as shown by a two years study at Massachusetts General Hospital. *Anesth. & Analg.*, Nov.-Dec. 1934.

67. Kanavel, A. B., and Koch, S. L. Preoperative preparation and postoperative care of surgical patients. *Bull. Am. Coll. Surg.*, 11: 14, 1927.

68. Lambret and Driessens. Postoperative hyperglycemia. Quoted in *J. A. M. A.*, 107: 294, 1936.

69. Lee, W. E. The present status of postoperative pneumopathies. *Prog. Med.*, 4: 351, 1931.

70. Lewis, D. Postoperative treatment. *Boston M. & S. J.*, 194: 913, 1926.

71. Maddock, W. G., and Coller, F. A. Water balance in surgery. *J. A. M. A.*, 108: 1, 1937.

72. Maddock, W. G., Boyden, A. M., and Malcolm, R. L. Mercresin, a new surgical germicide. *Am. J. Surg.*, 34: 47, 1936.

73. Mahlo. Relations between stomach and blood picture. *Deutsche med. Wchnschr.*, 61: 1358, 1935.

74. Mann, F. C., and Essex, H. E. The present status of the problem of traumatic shock. *Am. J. Surg.*, 28: 160, 1935.

75. Mason, J. T. Intravenous fluids; practical considerations in shock, dehydration, starvation, etc. *Industrial Med.*, 5: 168, 1936.

76. Matas, R. Continued intravenous "drys" with remarks on the value of continued gastric drainage and irrigation by nasal intubation with the gastric duodenal tube (Jutte) in surgical patients. *Ann. Surg.*, 79: 643, 1924.

77. Mensing, E. H. Treatment of intestinal obstruction involving problems of abnormal physiology and chemistry. *Wisconsin M. J.*, 33: 187, 1934.

78. Mettenleiter, M. W. Autohemotransfusion in preventing postoperative lung complications. *Am. J. Surg.*, 32: 321, 1936.

79. Moon, V. H., and Morgan, D. R. Shock: Mechanism of death following intestinal obstruction. *Arch. Surg.*, 32: 776, 1936.

80. Moynihan, B. Abdominal Operations, Chapt. 1. Philadelphia, 1926. W. B. Saunders.

81. MacCallum, W. G., Lintz, J., Vermilye, H. N., Liggett, T. H. and Boas, E. The effect of pyloric obstruction in relation to gastric tetany. *Bull. Johns Hopkins Hosp.*, 31: 1, 1920.

82. McNealy, R. W., and Willems, J. D. The absorption of glucose from the colon. *Surg., Gynec. & Obst.*, 49: 794, 1929.

83. McVicar, C. S. A discussion of the clinical and clinical laboratory findings in certain cases of obstruction in the upper gastrointestinal tract. *Am. J. M. Sc.*, 169: 224, 1925.

84. Ochsner, A. and Garside, E. Peritoneal adhesions. *Surg., Gynec. & Obst.*, 54: 338, 1932.

85. Ochsner, A., and Storck, A. Prevention of peritoneal adhesions by papain. *Ann. Surg.*, 104: 736, 1936.

86. Orr, T. G. Preoperative and postoperative treatment of gastric disease. *Am. J. Surg.*, 29: 26, 1935.

87. Ort, M. Treatment of postoperative acidosis by means of insulin. *Arch. Prov. de Chir.*, 27: 994, 1924.

88. Overholt, R. H. Postoperative pulmonary hypoventilation. *J. A. M. A.*, 95: 1484, 1930.

89. Pack, G. T. A modification of the Thalhimer apparatus for the slow intravenous injection of glucose and saline solutions. *J. Lab. & Clin. Med.*, 11: 1094, 1926.

90. Persson, M. Final results of gastric resections for cancer. *Ann. Surg.*, 86: 321, 1927.

91. Rehn, E. Operative shock. *Arch. f. klin. Chir.*, 177: 360, 1933.

92. Ryan, T. J. Hyperventilation in abdominal surgery. *J. A. M. A.*, 107: 267, 1936.

93. Shambaugh, P. Peritonitis as a factor in mortality of gastrointestinal surgery. *Ann. Surg.*, 104: 382, 1936.

94. Terry, A. H. Diabetic regime in surgical cases. *New York State J. Med.*, 35: 150, 1935.

95. Thalhimer, W. Insulin treatment of postoperative (nondiabetic) acidosis. *J. A. M. A.*, 81: 383, 1923.

96. Turner, G. G. What surgery offers in cancer of the stomach. *Glasgow M. J.*, 116: 137, 1931.

97. Walter, C. W. Preparation of safe intravenous solutions. *Surg., Gynec. & Obst.*, 63: 643, 1936.

98. Walters, W., Myerding, H. W., Judd, E. S. and Wilder, R. M. Surgery in diabetes. *Minnesota Med.*, 17: 517, 1934.

99. Walton, James. Carcinoma of the stomach. *Lancet*, 230: 1101, 1936.

100. Young, T. O. and Fowler, L. H. Postoperative management following gastric operations. *Minnesota Med.*, 7: 730, 1924.

RATIONAL POSTOPERATIVE TREATMENT FOLLOWING ABDOMINAL OPERATION

LOUIS E. MAHONEY, M.D., F.A.C.S.

Attending Surgeon, Santa Monica, Los Angeles General and St. Vincent's Hospitals

SANTA MONICA, CALIFORNIA

FOR all practical purposes, the average patient who undergoes an abdominal operation in the ordinary hospital today suffers the same amount of postoperative pain and distress and is subjected to approximately the same postoperative routine as prevailed twenty-five years ago. Judging from a study of textbooks, surgical literature and the "standing orders" of numerous hospitals, there seems to be an almost universal belief among our profession that the performance of a laparotomy brings in its wake a chain of symptoms which cannot be avoided and an amount of suffering which cannot be alleviated. Even very recent articles on the subject of postoperative management emphasize the assumed necessity of withholding water from patients and caution against the giving of food until late in the period of convalescence.

Likewise once or twice yearly some new drug is introduced having for its purpose the early reëstablishment of peristalsis and the prevention or amelioration of gas pains. The very fact that an enormous amount of research on the subject of intestinal motility has been done and that very praiseworthy results have been accomplished in the synthesia of drugs which will stimulate peristalsis is prima facie evidence that paresis of the small bowel with resultant abdominal distention does follow most operative maneuvers and that the usual and ordinary methods instituted for relief fail to accomplish the desired purpose. For the most part, attention has been directed to treatment of these conditions after their onset and but little thought has been applied to means of prevention. It is presupposed by most surgeons that the biochemical and physiologic processes of the postoperative patient undergo a very profound dislocation, and that this abrupt change requires for its correction very special, highly technical and for the unfortunate individual extremely distressing treatment. The lessons learned by medical men twenty years ago in the treatment of typhoid fever, for instance, are entirely disregarded, and for some obscure reasons, tradition and innate conservatism perhaps, are deemed entirely inapplicable to the handling of human beings after the usual run of surgical procedures.

The postoperative regime now in force in the great majority of American hospitals and approved by the bulk of surgeons is a heritage from the days of vigorous purgation, starvation and profound ether anesthesia. Perhaps twenty-five or thirty years ago when no reliable anesthetic agents save ether and chloroform were available, when preoperative preparation consisted of starvation plus castor oil, when every deep ether anesthetic was followed by from twenty-four to forty-eight hours of severe nausea and vomiting, when abdominal distention and severe gas pains followed all operative procedures within the abdomen, there may have been valid reasons for the drastic purgation, starvation or semi-starvation and frequent rest-disturbing enemata which followed every operation. However, in these modern times, with knowledge of proper, scientific preoperative preparation widely disseminated among the profession, with the wide choice of effective anesthetics and a better understanding of their uses and limitations, coupled with universal acceptance of the value of gentle, delicate, well planned technical maneuvers, there are no logical reasons for denying the unfortunate pa-

tient the opportunity to profit from this accumulated knowledge and to enjoy a smooth, uneventful convalescence.

While the hospital stay will probably never be exactly pleasurable, surgical progress together with humane feeling demand that the recovery period be so managed that an indelible impression of misery and suffering is not recorded on the patient's memory. Considering the matter from the purely practical and self-interested viewpoint of the surgeon who desires to extend the ministration of his art to the greatest possible number of persons requiring his care, there can be no reasonable doubt that accounts of severe pain and discomfort which have been widely spread among the lay public have prevented or postponed many badly needed operations and indirectly brought about numerous untimely deaths.

Severe criticism of long established customs and usages is not justified unless alternative and perhaps superior methods are suggested in their places. It is our intention, therefore, to set up certain standards by which good postoperative treatment may be measured and to discuss means by which, to our own satisfaction at least, we have been able to approach these objectives. For purposes of discussion, the following fundamental criteria are suggested:

1. There should be very little postoperative pain.

2. No abdominal distention or gas pains should be present.

3. Peristalsis should be quickly reëstablished.

4. The patient should regain entirely or closely approximate his preoperative weight before leaving the hospital.

5. There should be early restoration of muscular vigor and economic self-sufficiency.

With modern methods and particularly by careful attention to proved principles of normal physiology, it is now perfectly feasible to so manage the ordinary laparotomy that the main complaint of the patient during convalescence will be the pain and discomfort directly referable to the incision. Needless to say when the patient is troubled only by the comparatively unimportant factor of soreness in the recent wound, the surgeon is relieved of many anxieties which ordinarily harass him and disturb his peace of mind for the first few days following any surgical procedure. Incidentally much of the soreness in the ordinary wound can be avoided if heavy tension sutures are not used and interrupted black silk is employed for fascial closure, as heavy stitches invariably cut their way partially through skin and muscle and are a fruitful source of pain.

Temperaments and conditions vary and the surgeon must ever be ready to meet situations as they arise and to discard preconceived ideas. Always bearing in mind that human beings are not standardized and that the most carefully thought out routines frequently fail in their application to sick individuals, our plan is about as follows.

Water, hot, cold or from the tap, according to the patient's taste, is given immediately after consciousness returns and in such amounts as may be desired. Likewise a high caloric, protein-rich diet is advised very early. With gentle technical maneuvers and modern anesthetics, nausea and vomiting are becoming very rare, and as a result of these facts, a tray is presented the evening of the operative day and the patient is urged to eat some bread, toast, wafers, jello, breakfast foods or similar easily digested articles. Slight nausea and moderate vomiting are not feared as these function as automatic gastric lavage and obviate the necessity for use of Levine tubes, continuous suction, etc.

When the anesthetic employed has been ethylene cyclopropane or spinal, water is usually permitted orally within thirty minutes of the time patient returns to his room. With the use of ether by the open drop method, the ordinary individual will call for and retain water by mouth within one to three hours. Enough morphine or

pantopon is allowed during the first two or three days to prevent undue pain. Following the first night, a sedative selected from the barbital group is prescribed for oral use to produce sleep and quiet restlessness. The barbiturate, of course, reduces the amount of opiate required.

Fruit juices, and in particular orange juice, are sternly prohibited as it is well known that they possess little caloric value and unquestionably tend to produce excessive amounts of intestinal gas. Fine and Levenson have clearly shown that of all the commonly employed foods which form a part of the ordinary hospital liquid diet, orange juice and milk will form the largest amount of flatus. At no time is the usual hospital liquid diet prescribed as we believe that such a menu furnishes an insufficient amount of fluid, contains but very little energy measured in calories, and in our experience always leads to the production of abdominal distention with accompanying gas pains.

The fluid intake and output should be carefully estimated as considerably more water is needed to maintain systemic balance than is generally realized. Various workers have computed the loss through perspiration and respiration as somewhere between 1,000 and 2,000 c. c., and the urine and bowel discharges will account for a similar amount. Eminent physiologists and clinicians have asserted that the intestinal juices in the normal adult will reach the imposing volume of 3,000 to 4,000 c. c. every twenty-four hours. During the operation itself somewhere between 500 and 2,000 c. c. is lost through sweat, through the breath and in the bleeding which occurs. This figure depends, of course, upon the duration of the anesthesia and the severity of the operative interference. It, therefore, must be remembered that during the first day or two, if possible, the amount lost in the operating room plus the normal requirements of the adult must be supplied, and in actual bedside practice, this means that at least 3,000 c. c. must be ingested with the food or as plain water. As

a matter of clinical observation, it is fairly well established that the coöperative and intelligent patient will quickly reëstablish his water balance if allowed access to a pitcher and glass and permitted to drink the amounts which his system demands.

Besides water, large amounts of chlorides are lost on the operating table and in the bodily discharges, and the systemic reserve of glucose is depleted. Insofar as the chlorides are concerned, their diminution is compensated for through intake in the food with subsequent retention, while the glucose requirements are met through the ingestion of readily assimilable carbohydrates. Since the normal channels for the reception of these items are utilized, their assimilation and retention is more effective than if they are introduced by unnatural routes.

The early ingestion of easily handled solid foods plus ample water makes it unnecessary, except under unusual circumstances, to prescribe glucose and saline solutions by the subcutanious, intramuscular and intravenous routes. Avoidance of the pain caused by the insertion of needles for hypodermoclysis or venoclysis is greatly appreciated by the patient and, by adding to the number of hours he may rest in comparative comfort, greatly assists in his earlier recovery. Likewise it should never be forgotten, as has been shown by Fantus, that fluid given by mouth exerts a much greater diuretic effect than when administered intravenously. The natural channels for the reception of food and water are incomparably the most efficient. According to many authors the incidence of venous thrombosis and pulmonary embolism has doubled in the past twenty years and it is reasonable to assume that much of this increase is due to the more or less routine employment of hypertonic solutions.

The subject of intestinal gas has long interested surgeons and physiologists and many theories have been propounded to explain its occurrence. Certainly it is generally agreed upon that both in health and disease considerable amounts of air,

nitrogen, carbon dioxide, ammonia and methane are found in the bowel. There is no unanimity as to their origin as many writers feel that the air is swallowed, others call attention to the well known exchange of gases between the blood and intestinal mucosa with the comment that this is a two-way process, while still others blame fermentation and putrefaction resulting from bacterial action. Regardless of theoretical considerations, the facts remain that large amounts of gases form in health and much larger amounts when there is interference with intestinal motility from various causes. The combination of starvation plus operation is the one with which we are most vitally concerned at present.

In our early attempts fifteen years ago to feed patients early after operations we were greatly troubled by the problem of intestinal gas and abdominal distention. The first success attended our efforts after we banished the pitcher of orange juice which was formerly part and parcel of every hospital room immediately after operation. This simple maneuver produced encouraging results. Since that date excellent scientific work, in particular that of Fine and Levenson, has shown that orange juice is readily fermentable and produces truly enormous quantities of gas. My attention was then directed to the clinical observation that healthy persons who go on reducing diets almost always select what really amounts to an ordinary, hospital liquid diet and that they invariably suffered from gaseous distention. The next step was to eliminate entirely the liquid diet and to prescribe solid food at once. Further diminution of gaseous distention and increased comfort was obtained. However, it was still necessary to make use of pituitrin and other drugs and to employ frequent enemata in order to stimulate peristalsis.

From observation of the practices of internists in their handling of colonic stasis, it was assumed that an adjuvant which would furnish bulk, supply lubrication and possess laxative qualities would be highly desirable. Mineral oil in agar, with or without the addition of small amounts of phenolphthalein best suited these specifications and has been employed routinely in 1 ounce doses three times a day whenever tolerated. As all clinicians are aware, there are some few individuals who will not or cannot take mineral oil. In the event the patient registers no objection to the oil and agar mixture, the bowels will move easily and without appreciable distress on the afternoon of the third or the morning of the fourth day. Rarely will enemata be necessary and small amounts of flatus which may develop are readily taken care of by means of the rectal tube. Once the first bowel action has occurred, the dosage of the oil and agar mixture can be gradually reduced to the point where one or two movements take place daily without troublesome leakage. Immediately following the first action of the bowels, the hospital general diet is prescribed with the idea in mind of restoring the weight level to the same figure, or at least very close to it, as on the day of admission.

Free and unrestricted movements in bed are likewise quite helpful as they tend to promote good circulation thus diminishing the possibility of emboli, and most important of all, assist in restoring the patient's self-confidence. Enemata are but rarely required or employed as they tend to wash out the normal protective coating of mucus on the lumen of the bowel and thus probably aid in producing colonic stasis with accompanying gas formation. It is even possible that instances of colitis of varying grades of severity may be attributable to the injudicious use of irritant enemata during convalescence.

Most patients receive far too much treatment and over-solicitous attention. Rest is the most valuable weapon in the surgeon's armamentarium and the most often overlooked. The normal reparative mechanism of the human body is truly amazing and should be permitted to work its miracles unhampered by unnecessary or even harmful therapeutic maneuvers. Nurses need careful and tactful instruction,

if the above outline régime is in force, as from past experience and training they are likely to feel that they are not doing their full duty unless both they and the patient are kept very busy.

The type of management described has been applied to appendectomies, cholecystectomies, stone in common duct, early mechanical obstruction, herniotomies and various types of pelvic surgery. It has not been used in severe intestinal obstruction, major gastric operation, malignancies, general peritonitis, and is not indicated when abdominal distention is present when the patient is first seen by the surgeon. However, as mentioned before, the plan is applicable to by far the majority of operations performed by the trained individual. The method will probably fail in the hands of the surgical tyro or occasional operator because of faulty preoperative preparation, rough handling of tissues or improper evaluation of the surgical risk involved. So far the patients, for the most part, have been first class surgical risks.

Approximately 350 patients have been given the above described dietary management and sixty-eight have been given mineral oil emulsion. There were four instances in which the mineral oil therapy had to be abandoned.

The advantages accruing were the comparative freedom from pain, absence of distention or gas pains, increased cheerfulness, excellent appetite, and most important of all, the early reëstablishment of normal weight and quick return to ordinary duties. Several individuals actually gained weight while in the hospital. Avoidance of the use of glucose and saline solutions appreciably lowered the hospital costs and thus relieved economic distress. A great deal of the present agitation with regard to high hospital costs can be allayed by general adoption of these methods. Less nursing is likewise needed and this also does wonders to help the financial situation.

In the course of our investigations, it was observed that several patients lost very little weight following major surgical procedures, and about two years ago it was determined wherever possible to record accurately the pre- and postoperative weights. While it was not always feasible to weight all patients since some operations are emergencies, some patients returned home before becoming ambulatory, etc., we now have accurate observations on some forty-three individuals. A list of these is given below

Operation	No.	Gained	Lost	No Change	Average Loss, Lb.
Appendectomy	17	3	13	1	3
Cholecystectomy	10	2	7	1	2½
Salpingectomy	3		3		3
Herniotomy	3	1	1	1	½
Common duct obstruction	1		1		3
Uterine suspension	1	1			2
Duodenal ulcer ruptured	1		1		4½
Hysterectomy	7		7		2½
Laparotomies	43	7	31	3	

There are no statistics in the literature, at least none which were available to us, showing the average loss of weight following abdominal operations, but it is our impression that the decrease under usual methods of management is somewhere between 7 and 15 pounds.

There were four failures of this mode of treatment either through inability to take mineral oil or lack of appetite. One man who refused to take mineral oil emulsion and would not eat until the sixth day after the removal of a midly inflamed appendix lost 17 pounds. The wife of a physician who desired a reduction in weight, without our knowledge deliberately selected foods of low caloric value and showed a loss of 8 pounds on the tenth postoperative day. However, there was no distention or gas pain. Two rather nervous young women who underwent pelvic surgery refused to eat or to take mineral oil.

Table 1 shows the details of management of the forty-three patients.

TABLE I

Age	Sex	Operation	Anesthetic	No Ene-mata	Day Bowel Moved	Weight Preop.	Weight Postop. Day	Weight Postop. Pounds	Loss	Gain
30	F	Appendectomy / Removed ovarian cyst	Cyclopropane	n	3	120	10	122	n	2
38	M	Appendectomy	Ether	o	3	180	13	176	4	0
44	M	Appendectomy	Ether	n	3	192	11	187	5	0
50	F	Cholecystectomy	Ethylene	o	4	142	14	142	0	0
42	M	Appendectomy	Ethylene	o	3	220	7	222	0	2
47	M	Explored bile ducts / Freed adhesions	Ether	2	5	225	2	222	3	0
44	F	Cholecystectomy	Ethylene	o	3	132	10	136	0	4
50	M	Appendectomy	Ethylene	o	3	162	10	160	2	0
38	F	Hysterectomy / Supravaginal cervical cauterization	Ethylene	o	4	114	11	111	3	n
50	F	Appendectomy	Ethylene	o	3	136	10	135	1	0
52	F	Cholecystectomy	Ethylene	o	4	154	14	149	5	0
39	F	Ruptured pyosalpinx / Uterine fibromyoma	Ethylene	1	3	146	12	145	1	n
42	F	Hysterectomy / Supravaginal cervical cauterization	Ethylene	o	4	138	12	136	2	0
20	F	Appendectomy	Ether	o	3	118½	14	115½	3	0
28	M	Appendectomy	Ether	o	3	158	13	160½	0	2½
4	M	Ferguson hernia repair / Jaboulay hydrocele operation	Ether	o	3	39	8	38	1	0
55	F	Cholecystectomy	Ethylene	o	4	116	12	112	0	4
24	F	Appendectomy	Ethylene	o	3	157	6	156	0	1
28	F	Appendectomy	Ethylene	o	3	150	11	152	2	0
4	M	Ferguson hernia repair	Ether	o	3	45	14	45	0	0
43	M	Appendectomy / Freed colonic adhesions	Ethylene	o	3	138	9	141½	3½	0
35	F	Appendectomy / Supravaginal hysterectomy	Ethylene	o	4	238	10	236	0	2
43	F	Studdiford perineoplasty / Supravaginal hysterectomy / Cervical cauterization	Ethylene	1	5	125	13	122½	0	2½
22	F	Appendectomy / Salpingectomy right / Ovarian cyst right	Ethylene	o	4	116	14	112	0	4
11	M	Appendectomy	Ether	2	5	82	10	82	0	0
56	F	Cholecystectomy	Ethylene	o	3	125	9	122½	0	2½
6	M	Bassini hernia repair / Circumcision	Ether	o	3	53¾	4	54	¼	0
38	M	Appendectomy	Ethylene	o	4	158	6	154	0	4
55	M	Perforated duodenal ulcer	Ethylene	1	3	160	9	155½	0	4½
61	F	Cholecystectomy	Ethylene	1	4	177	10	174	0	3
37	F	Appendectomy	Ethylene	1	4	136	11	132	0	4
59	F	Cholecystectomy	Ethylene	3	5	153	10	150	0	3
23	F	Appendectomy Baldy-Webster uterine suspension	Ether	o	3	125	10	124	0	1
28	F	Removal of ovarian cyst	Ethylene	o	4	118	10	120	2	0
50	F	Cholecystectomy	Ether	o	4	155	9	155½	½	0
38	F	Cholecystectomy	Ethylene	o	4	127	11	125	0	2
31	F	Supravaginal hysterectomy	Ethylene	o	4	104	8	101½	0	2½
38	F	Supravaginal hysterectomy	Ethylene	o	4	114	10	110	0	4
50	F	Supravaginal hysterectomy	Ethylene	o	4	168	10	164	0	4
24	F	Salpingo-oophorectomy right / Salpingectomy left / Appendectomy	Ether	o	5	106	8	101	0	5
35	F	Bilateral large chocolate ovarian cysts	Ethylene	o	4	124	10	122	n	2
43	F	Cholecystectomy	Ethylene	o	3	153	9	150	o	3
28	M	Appendectomy	Ether	o	2	148	6	144	0	4

SUMMARY

Present day postoperative treatment leaves much to be desired.

Fundamental criteria of good management are suggested.

A simple routine is suggested. Early feeding of solid food with high protein and caloric value has been used in over 350 laparotomies.

Mineral oil with phenolphthalein has been employed sixty-eight times with four failures.

Forty-three patients whose weights could be carefully checked show an average loss of approximately 3 pounds. Seven patients

actually gained weight and three showed no change.

REFERENCES

1. ALVAREZ, W. C. Is purgation justified? *Surg., Gynec. & Obst.*, 26: 651, 1918.
2. ALVAREZ, W. C. Postoperative gas pains. *California State J. Med.*, 16: 338, 1918.
3. MAHONEY, L. J. Action currents in stomach and intestine. *Am. J. Physiol.*, 58: 421–430, 1922.
4. TAYLOR, F. S., TERRY, W. J., and ALVAREZ, W. C. Preoperative and postoperative care. *J. A. M. A.*, 79: 1578, 1922.
5. CUNNINGHAM, S. P. Cause and prevention of gas pains. *Anesth. & Analg.*, 7: 376, 1927.
6. FANTUS, BERNARD. Fluids postoperatively. *J. A. M. A.*, 107: 1, 1936.
7. HIBBARD, J. S. Gaseous distension associated with mechanical obstruction of intestine. *Arch. Surg.*, 33: 146, 1936.
8. KANTOS, J. S., and MARKS, J. A. A study of intestinal flatulence. *Ann. Int. Med.*, 3: 403, 1929.
9. OTTENHEIMER, E. J. Gas pains. *New England J. Med.*, 213: 608, 1935.
10. BONNEY, V. Functional derangement of intestine that follows abdominal operations. *Lancet*, 2: 1323, 1933.
11. FINE, J., and LEVENSON, W. S. Effect of fluids in postoperative distention. *Am. J. Surg.*, 21: 184, 1933.
12. VOGEL, K. Method for preventing intestinal inflation following operations. *Zentralbl. f. Chir.*, 59: 1018, 1932.
13. ORR, THOMAS G. Therapy of abdominal distension. *J. Kansas M. Soc.*, 33: 3, 1932.
14. CUTTING, R. A. Abdominal distension and gas pains. *Am. J. Surg.*, 12: 561, 1931.
15. MELCHIO, E. Treatment of gastro-intestinal atony and paralysis. *Chirurgie*, 1198, 1929.
16. COUNTRYMAN, G. W. Postoperative enemata. *Northwest. Med.*, 33: 355, 1934.
17. KAHN, R. M. *Bulletin Am. College of Surgeons*, 76, April, 1937.
18. TORREY, J. C. Regulations of intestinal flora of dogs through diet. *J. M. Research*, 39: 415, 1919.
19. HERRIN, R. C., and MEEKS, W. J. Distension as a factor in intestinal obstruction. *Arch. Int. Med.*, 51: 152, 1933.
20. McIVER, M. A., BENEDICT, E. B., and CLINE, J. W. Postoperative gaseous distension of intestine. *Arch. Surg.*, 13: 588, 1926.
21. COLLER, F. A., and MADDOCK, W. G. Dehydration attendant on surgical operations. *J. A. M. A.*, 99: 11, 1932.
22. STEWART, J. D. Fluid therapy in surgery, a critical review. *New England J. Med.*, July 9, 1936.
23. COLLER, F. A. DICKS, V. S., and MADDOCK, W. G. *J. A. M. A.*, 107: 1522, 1936.
24. CABOT, H. Water. *Surg., Gynec. & Obst.*, 63: 681, 1936.
25. WOLFER, J. A. Nutritional status of the surgical patient. *Surg., Gynec. & Obst.*, 63: 607, 1936.
26. MADDOCK, W. G., and COLLER, F. A. Water balance in surgery. *J. A. M. A.*, 108: 1, 1937.
27. THOMPSON, W. D., ROWDIN, J. S., and IRVING, F. L. Effect of hypoproteinemia on wound disruption. *Arch. Surg.*, 500–508, 1938.
28. ORR, T. G. The use and abuse of intravenous therapy in surgery, *J. Missouri State M. Soc.*, 34: 219, 1937.

SURGICAL CONSIDERATIONS OF TEMPERATURE IN LIGATED LIMBS

FREDERICK M. ALLEN, M.D.

NEW YORK CITY

SEVERAL recent papers have described certain phases of the writer's observations on local asphyxia produced by applying rubber tourniquets to various parts, particularly the limbs, of several warm-blooded species.[1] Locally, the tissues are able to tolerate much longer periods of absence of circulation than supposed by the average surgeon. The systemic effects constitute an ideal form of experimental secondary shock, which can be fairly accurately standardized in different grades of severity according to the mass of tissue ligated and the length of time. By this method, indirect evidence was obtained which seems to favor the toxic theory of the causation of secondary shock. The present paper will add observations on the modifying influence of temperature, with suggestions concerning possible applications in surgery.

SURVIVAL TIME AT ROOM TEMPERATURE

Experiment. The right foreleg of a dog was tightly ligated a short distance above the wrist for fifteen hours at room temperature. Light amytal narcosis was used at the beginning, when the tourniquet was obviously a source of discomfort. By the time the narcosis passed off the ligated part of the leg was cold and the dog paid no attention to it, so that he could be left free and unmuzzled; the animal ate and drank plentifully. The leg became hot and swollen within fifteen minutes after release. It was paralyzed and without sensation. Within a few hours the edema extended above the elbow, and there was slight blood-tinged oozing through the skin between the toes. All these effects subsided during the following days, without ulceration or permanent disability.

A blood count just before release of the tourniquet showed red cells numbering 6,350,000. A second count at the height of edema three hours after release showed: red cells 5,300,000, leucocytes 31,000. The ligation of a forefoot for fifteen hours was thus unable to produce constitutional symptoms of shock, and the edema of one foreleg failed to cause concentration of the blood in a strong dog able to eat and drink normally.

Wilson and Roome,[2] who took special care to assure complete stoppage of circulation, found no gangrene in dogs' legs after as long as twenty hours of ligation. Their animals did not live long enough for them to make sure that the legs might not have been lost secondarily through thrombosis or ulceration at the zone of ligation, though the tissues distal to the ligation zone seem able to endure probably more than fifteen hours. The wing of a chicken has survived nine hours of ligation, and the actual limit may be still longer. Among other variables, the activity of tissue metabolism may be one cause of differences in resistance to asphyxia in different species. This factor must also be affected by temperature.

LIGATIONS AT ELEVATED LOCAL TEMPERATURES

Unanesthetized rats were comfortably immobilized, with a ligated hind leg projecting through a hole in a board into a water bath or hot air chamber. The close fitting of the leg in the hole and other precautions prevented any appreciable heating of the body, and the absence of fever was proved by frequent rectal temperature readings. The temperature of the water or air chamber was regulated within

a range of approximately one degree during the experiments.

Accepting 47°C. as the approximate temperature of beginning heat coagulation in mammalian muscle, and considering the probability that the interior temperatures of the leg were not quite so high as that of the bath, the experiments were begun with water between 46° and 47°C. One hour of such ligation resulted in the death of rats within one hour after release. Ligations of only fifteen to thirty minutes sufficed to cause gangrene of the leg, with or without subsequent death of the animal.

A series of experiments was then performed with descending gradations of temperature. On account of the two variables determining survival, namely temperature and time, the number of such experiments had to be large. The greatest obstacle to precision was encountered in a third variable, namely the different resistance of different portions of the leg. The feet, and particularly the toes, were much more sensitive than the upper leg, evidently because of the lack of hair, the thin delicate skin and the smaller diameter, permitting freer penetration of heat. Therefore vesication and sloughing beginning in the toes or feet sometimes extended upward so as to cause final loss of the leg, although the upper tissues seemed to have survived the primary damage. A modified procedure—coating the feet with some protective substance—was not carried to any consistent conclusion. The results obtained with the unmodified method were as follows:

In water at 45°C., ligation of the entire hind leg for thirty minutes was fatal. These deaths occurred within two or three days, without gangrene or stripping of the skin or hair, but with bright pink hyperemia of the foot and large edema extending up into the flanks. Ligations below the knee, which did not cause fatal shock, were tolerated by the local tissues nearly up to one hour. Periods between one and one and one-half hours caused necrosis, usually developing gradually in the course of a day or two and ending in spontaneous amputation or death.

In water between 42 and 43°C., fatal shock developed after one and one-half hours, and with lower ligations the survival limits of tissues were about the same.

In water between 40 and 41°C., fatal shock could be produced in two to two and one-half hours, and with lower ligations the survival limits of tissues were about three to three and one-half hours.

In water between 38 and 39°C., fatal shock was produced within two to two and one-half hours. With lower ligations the survival limit for tissue was not established positively, but was probably about four hours.

The results with warm air were less satisfactory, presumably because of the poorer penetration of this form of heat. The tolerance limits were not accurately established, but were always reduced to a fraction of the known limits at room temperature.

In representative animals, the supposition that the cause of the acute deaths was secondary shock was tested by blood counts during the ligation and subsequently up to death. The red cell counts were found to rise by two to three million, while at the same time the emptiness of visible blood vessels indicated a diminished blood volume. The rectal temperature during ligation was usually 99 to 100°F. in quiet animals, and exceptionally rose to a maximum of 101.2 with struggling or other disturbances. Development of shock was accompanied by the rapid extreme fall of temperature which is particularly characteristic of small animals—down to 94–96 in the mild degrees which ended in recovery, and down to 90 or lower in severe fatal cases.

It is astonishing to find such destructive effects of slight elevations of temperature, in a water bath which feels only comfortably warm to the hand. The appearances rather strikingly resemble ordinary burns; particularly, the erythema, vesication and necrosis of limbs asphyxiated for

a short time a little above 40°C. give an appearance which is scarcely distinguishable from that of normal limbs dipped briefly into boiling water. That this resemblance is more than superficial is indicated by clinical experience of the ease with which tissues of poor vitality or circulation are burned at comparatively low temperatures. The writer witnessed one case of an arteriosclerotic diabetic whose feet were severely blistered in a Turkish bath which felt comfortable to him and his companions. Temporary ligation of limbs is one standard method of producing shock, and burns are another well-known method. It is interesting that the two methods meet in these experiments, and that their effects in producing both local tissue changes and constitutional shock become added to one another.

Normal tissues have a considerable protection against heat in their blood flow, which acts as a cooling system. That this is not the chief explanation is shown by the fact that asphyxiated tissues show rapid necrosis at temperatures well within the range of fever which normal tissues can tolerate for days or weeks. Evidently the raised temperatures, by accelerating local metabolic or autolytic processes, create increased needs for a supply of oxygen and food and for removal of toxic waste products, and, without circulation, tissue death is thus hastened. In harmony with this interpretation is the behavior of the nerves, which degenerate more rapidly (as judged by paralysis and anesthesia) at elevated temperatures than at room temperature.

One school of investigators finds a satisfactory mechanical explanation of shock in the vasodilatation and fluid exudation resulting from both asphyxial and thermal injuries. Obviously, the accumulation of toxic substances in the ligated tissues, occurring at increased rates at higher temperatures, harmonizes equally well with the chemical or toxic hypothesis.

LIGATIONS IN FEBRILE ANIMALS

Rats, comfortably immobilized in individual well-ventilated boxes, were placed thus in a warm-air chamber. Immobilization was necessary because muscular activity was found to be the chief cause of irregular temperatures and dangerous hyperpyrexia. Brief removal from the chamber every fifteen minutes for taking rectal temperatures did not noticeably alter the course of the fever. At the end of the experiment, milk was fed or 10 c.c. of physiologic saline was injected subcutaneously. Rectal temperatures between 104° and 106°F. could be tolerated without danger for about one hour, or 103° for three or four hours. The oven temperatures for this purpose were mostly between 33° and 35°C. The rectal temperature always fell to normal or subnormal levels within one-half to one hour after removal from the oven. The results with this crude method were too variable for accurate standardization, and there was only the general demonstration that the limits of tolerance, as regards both survival of the local tissues and constitutional shock effects, are much shorter than when the legs are ligated at room temperature.

LIGATIONS AT REDUCED TEMPERATURES—SHOCK

Unanesthetized rats were prepared in practically the same way as for the experiments with local heat, and the ligated legs were immersed in ice-water to a level barely above the tourniquet. Precautions were used to prevent wetting the body and to maintain body warmth. By occasional stirring, the mixture of ice and water immediately adjacent to the leg was kept at approximately 2°C.

Mention has been made elsewhere of the reduction or abolition of shock under these conditions. For example, the high ligation of one hind leg for five hours at room temperature is fatal, but the leg can be safely ligated for more than eight hours in ice-water. The high ligation of

two hind legs for three hours is fatal at room temperature, but in ice-water periods of more than five hours are easily tolerated. The stoppage of aortic circulation by a tourniquet around the lower abdomen for about one and one-half hours proves fatal at room temperature, but by refrigeration of the entire hind-quarters to a level just above the ligature, survival is possible after three hours or more. On account of poor facilities, it was impossible in some instances to prevent the rectal temperatures from falling even lower than in ordinary shock, sometimes to 28° or 27°C. But, contrary to the rule in shock, subsequent warming of the rat allows it to recover readily.

Under these conditions the red cell count is found either to remain approximately constant or to increase within moderate limits. The slightness of the swelling and exudation as compared with room temperature suffices to explain the prevention of constitutional symptoms according to the physical hypothesis. The inhibition of local metabolism furnishes an equally good explanation to upholders of the chemical or toxic hypothesis. It may be added that the nervous degenerations, manifested in paralysis and anesthesia, are much less than for an equal period of ligation at room temperature. If this fact is accounted for by retardation of metabolism in the nerves, the greater damage of motor as compared with sensory nerves still requires explanation.

SURVIVAL TIME AT REDUCED TEMPERATURE

Physiologists are familiar with the preservation of living tissues in the icebox for long periods, sometimes amounting to a number of months. More recently physicians have become acquainted with the preservation of blood by refrigeration for weeks or months, after which it can be used for transfusion like fresh blood. The present experiments were not suggested by the above facts, but grew out of the work with shock. Begun six years ago,

this phase of the work was interrupted by so many interfering conditions that it could only lately be carried to the point of publication. These are the first observations of the application of the principle of refrigeration to organs still connected with the animal.

This principle has been used to preserve the ligated legs of rats for periods up to twenty-four hours, with later recovery. Owing to the difficulty of maintaining the body temperature and strength in these small animals, the experiments were continued upon larger species. Other papers[3] describe ligations of the legs of rabbits, cats and dogs at points between the knee and ankle, with refrigeration at about 2°C., for various periods up to fifty-four hours. Similarly, the application of a tourniquet to the lower thigh has been safely endured for as long as twenty-four hours.

No attempt has been made to determine the actual time limits of survival of tissues under these conditions. In practice, the limit is not set by the tolerance of the refrigerated tissues for asphyxia but by the consequences of direct pressure in the zone of the ligature. Though these effects are much less than at room temperature, yet after the longest ligations ulceration commonly occurs in this zone.

The edema and inflammation which develop in the asphyxiated tissues are similar to the results of much shorter ligations at room temperature. The recovery also usually requires less time.

The nearer to the foot the tourniquet can be placed, the better for determining the true survival limits of the local tissues. Ligations above the knee can be tolerated only for a shorter time, and at higher levels in the thigh the periods become still shorter, though always much longer than at room temperature. The reason is obvious in the thickness of the thigh. There is a considerable mass of tissue in the interior, at and below the zone of ligation, which is kept more or less warmed by the adjacent tissues which

still retain circulation and therefore cannot be efficiently refrigerated by the ice-water outside the skin. Accordingly, both local gangrene and fatal shock must result with this crude method if the ligation is prolonged to a period, such as forty-eight hours, which can safely be tolerated with ligations in the thin part of the leg where the refrigeration is more efficient.

APPLICATIONS IN SURGERY

Before undertaking any clinical application of the principle, it would be well for surgeons to convince themselves by trials in animals that ligation and refrigeration for five hours or longer is not only harmless but also inhibits local tissue damage and general shock. Theoretically at least, the following possible uses may be suggested:

1. As mentioned elsewhere, this method has actually been employed for amputation in a few cases of diabetic gangrene. The use of a tourniquet, which has always been forbidden in such conditions, has not thus far created any trouble at the reduced temperature, even with badly sclerosed vessels. There has not yet been opportunity for a sufficient number of trials to decide the advantages and disadvantages as compared with ordinary anesthesia. The experience only proves that amputation can thus be performed in the human subject without pain or shock, and that the wounds can heal satisfactorily. It is hoped that the anesthesia of protoplasm may have some superiority over the mere anesthesia of nerves, and may be serviceable, especially in cases of bad operative risk on account of poor general condition. It must be emphasized, however, that the method is strictly in an experimental stage and its advisability is not assured.

2. For emergency purposes this plan can be recommended more positively. Occasionally there are accidents in isolated localities, and on a larger scale there are military emergencies, as for example recently in Spain or China, when anesthetics may be lacking or surgical care may be delayed. If ice is available, the tourniquet and refrigeration can be applied by anybody of reasonable intelligence. Hemorrhage and pain are thus stopped, and both the local tissue damage and the systemic shock which result from prolonged ligation at ordinary temperatures are abolished. Theoretically a limb might be preserved in ice-water or cracked ice for twenty-four hours or more, especially if the ligation is not too high, but it is safer to limit the time to five or six hours if possible. In this way an overworked military surgeon might, after attending to the most urgent cases of body wounds, find all his limb cases comfortable and ready; he could proceed with the necessary amputations or repairs without the delay of anesthesia or hemostasis, and after removal of the tourniquet could depend upon satisfactory circulation and healing.

3. Even under ideal conditions, there are instances in which a surgeon performing a plastic or other tedious operation may wish to keep a limb bloodless for several hours. As mentioned elsewhere, the unnecessary fears in regard to such a stoppage of circulation arise essentially from two causes: (1) paralyses and contractures may be caused by an improper tourniquet, i.e., one that is too tight, wide or inelastic; especially, a narrow rubber tube is less painful and less damaging than the broad compression which surgeons have favored without evidence; (2) all local tissue damage is accentuated in proportion as the temperature is elevated. A limb that is to be kept bloodless for any considerable period should therefore at least be chilled with icebags. Plastic and other results can be improved by the better preservation of tissue vitality.

4. When a limb is deprived of circulation through an accident, such as a wound or embolism, the procedure must depend upon the degree of deprivation. If there seems to be enough remaining blood supply to keep the tissues alive, the use of warmth to dilate the remaining vessels may be rational But if no such hope exists, as for example in blocking of the femoral artery,

it must be emphasized that the warming of the limb either with artificial heat or with bedclothes is the worst thing that can be done. Each hour that passes increases two dangers: (1) intravascular clotting due to damage of the endothelium by asphyxia; (2) systemic shock and intoxication if the circulation is restored. Therefore embolectomy, for example, is generally unsuccessful after longer than four or five hours, and this time is shortened by heat. On the other hand, as an inadequate collateral circulation probably does more harm than good, the best immediate treatment will be the application of a narrow tourniquet (such as a rubber tube) above the point of obstruction, and the immediate thorough chilling of the limb in ice or ice-water. Pain is thus stopped and other damage minimized. The time during which the human thigh can be constricted at ice temperature without danger of subsequent shock is unknown, but it is certainly much longer than at higher temperatures. The feasible period for arterial suture or embolectomy is correspondingly lengthened and the chance of success improved, on account of the vastly better preservation of vitality in the tissues and vessels. An anesthetic will be needed if the operation is early, but not if there has been time for thorough chilling of the limb. An important advantage is that the tourniquet and refrigeration can be applied, if necessary at home or by an attendant, thus forestalling the damage that usually occurs before the operation can be begun.

CONCLUSIONS

1. The survival limit of the leg tissues of several mammalian species, deprived of circulation at room temperature, is above fifteen hours.

2. As the temperature is progressively raised the effects of asphyxia are accelerated as regards production of both local gangrene and systemic shock.

3. With reduction of temperature, these effects are retarded. Near 0°C., recovery is possible after ligation for fifty-four hours, and under better conditions this time could perhaps be considerably extended.

4. According to the experimental evidence, the method of ligation and refrigeration may find some valuable applications in surgery of the limbs, especially under certain emergency conditions.

REFERENCES

1. ALLEN, F. M. The Tourniquet and local asphyxia. *Am. J. Surg.*, 41: 192–200, 1938. Effects of ligations on nerves of the extremities. *Ann. Surg.*, 108: 1088–1093, 1938. Physical and toxic factors in shock. *Arch. Surg.*, 38: 155–180, 1939.
2. WILSON, H., and ROOME, N. W. *Arch. Surg.*, 32: 334, 1936.
3. ALLEN, F. M. Local asphyxia and temperature changes in relation to gangrene and other surgical problems. *Tr.A.A.Physicians*, 52:189, 1937. Resistance of peripheral tissues to asphyxia at various temperatures. *Surg., Gynec. & Obst.*, 67: 746–751, 1938. Experiments concerning ligation and refrigeration in relation to local intoxication and infection. *Surg., Gynec. & Obst.*, June 1939.

MOBILIZATION AND IMMOBILIZATION*

AS APPLIED TO THE ANGLE PLATE IN THE TREATMENT OF FRACTURES

George W. Hawley, m.d., f.a.c.s.

WESTPORT, CONNECTICUT

THE writer recalls the interest aroused by Lucas-Championierre forty years ago when he advocated so forcefully mobilization in the treatment of fractures. In support of his ideas he pointed out the fact that fractures of the ribs healed with motion, at most restricted, but uninterrupted. Many unrecognized fractures recovered without effort to protect the injured part from motion, particularly before the introduction of the penetrating Roentgen ray. There followed much discussion between those who were inclined to favor mobilization and those who remained arch-advocates of immobilization.

Mobilization can hardly be called a method of treatment. The fact that fracture healing takes place in the presence of motion, shows what a good job nature can do in spite of motion, what a high-geared creature man is as a self-repairing machine, and that "fractures unite with treatment, without treatment and in spite of treatment." It is really surprising that delay of union, or failure of union, is not more common. Mobilization, per se, may reasonably well be dismissed as a method or principle of treatment.

IMMOBILIZATION

Emphasis has been put on the principles of fracture treatment, and justly so. "Principles never change" is an expression oft repeated. And immobilization has stood the test of time.

By immobilization we mean immobilization of the fracture, not immobilization of the limb, of which the bone is only a part. A whole limb is immobilized for the purpose of securing immobility of the broken bone at the site of fracture. It is easy to fall into the habit of thinking in terms of the limb, because much or all of a limb is immobilized in order to immobilize a fracture, and then imperfectly.

By mobilization is not meant mobility of the fracture, which Lucas-Championerre believed beneficial rather than otherwise, but mobility of the muscles and joints without movement or displacement of the fracture surfaces. This usually means waiting until healing has advanced to the point where it is safe to remove cast or splint and when the muscles have already lost much of their elasticity and weakly contract in response to nerve impulses, no matter how strong the mental effort.

Up to the present, observations on fracture repair have been made with the limb encased in plaster and with imperfect immobilization of the broken fragments. Motion between fracture ends takes place within casts. Muscle pull is sufficient to alter position of fragments and sometimes causes flat plates of the Lane type to bend or break.

Such is fracture healing with the limb in a state of physiologic rest. The picture is a familiar one: also the appearance of the limb after healing is complete. From this picture the pattern and the time of healing has been formulated. The duration of immobilization and the time required for fracture consolidation, for recovery of use of muscles and joints after consolidation, and the pattern runs true to form.

When much or all of a limb is immobilized for several weeks, repair of a fracture takes place with all the tissues in the retrogressive process of atrophy. The muscles shrink and lose, not only their contractile power, but their elastic prop-

erty. Joints stiffen, and the influx of arterial blood is lowered. There is lymphatic stasis.

FIG. 1.   FIG. 2.

FIG. 1. X-ray before operation. Arm in Thomas arm splint. Reduction diffi- cult to secure and retain.

FIG. 2. After operation. There is separation of the fragments which can be over- come by forcing fracture ends together before the last screws are introduced. Despite separation, repair was prompt. Four screws have been found suffi- cient in fractures of humerus, radius and ulna. In femur and tibia fractures six screws are used.

It is obvious that if a fracture can be effectively immobilized internally, there is little or no occasion for external immo- bilization. The principle does not change. The method is changed. And the physi- ologic conditions under which fracture repair takes place are changed. Healing takes place in the presence of active motion, if limited. Physiologic function is little interrupted, only reduced in degree. Mus- cles retain both their contractile and elastic properties. Joints continue mobile, and the arterial, venous and lymphatic flow approaches normal. Muscle action acts to pump in fresh blood and increase the influx of calcium.

Under such conditions there exist immo- bility of the fracture and, at the same time, mobility of the limb, with fracture repair taking place in the presence of active motion of muscles and joints, accustomed to daily exercise and activity, instead of in a state of immobility and inactivity.

The pity of it is, we know so little about the process of fracture repair. In con- versation with the writer during a visit to his hospital in Compiegne during the war, Carrel expressed the belief that when the process of fracture repair was better understood, fractures would unite in much less time than they do now. The thought was suggested that it might not be so much the need of methods or agents to stimulate fracture repair, as removing inhibiting factors which retard repair and the release of forces now held in check. At the time, Carrel's hospital was largely filled with war fractures.

A CASE ILLUSTRATING IMMOBILIZATION
OF THE FRACTURE AND MOBILIZATION
OF THE LIMB[1]

P. B., age 30, fractured the right humerus, September 21, 1938. Difficulty was experienced

[1] By courtesy of Dr. D. C. Patterson, Bridgeport, Connecticut. Poor health makes it necessary for the writer to depend on friends and others to put this plate into use. Dr. Patterson kindly asked me to see the operation and direct the after care.

in obtaining closed reduction, and at operation on September 28 the fragments were aligned. One fragment was lifted and a slot cut with a small Albee saw blade and widened by running the saw back and forth. One-half of the angle plate was secured to one fragment. A slot was then cut in the opposite fragment and the plate fitted into place.[1] Drill holes were made with a $5/64$ inch drill point through both cortices. The holes in the proximal cortex were enlarged with a carpenter's awl. Screws somewhat longer than the diameter of the bone were then introduced. They fitted tightly and a manual test showed strong immobilization of the fracture. After the wound closed, light temporary coaptation splints were applied from the axilla to the elbow.

Elapsed time of operation was one hour and five minutes. Complete rest was advised for eight days in order to limit the inflow of blood and favor the exit of exudate from the region of fracture. The accompanying pictures tell the rest of the story.

In this case[2] the internal immobilization was all that could be desired. The screws held. None became loose. The patient experienced no pain or sensation at the site of fracture. He had, and showed, confidence in the strength of the plated humerus. New bone appeared early in the roentgenographs and progressively increased.

The "principle" of immobilization calls for effective replacement and immobilization of the fracture. The more nearly perfect the immobilization, the closer we come to fulfilling the principle of immobilization. There may be more significance than we imagine in the difference between perfect and imperfect immobilization. *It is possible that the more "specific" the immobilization, the more active and positive the process of fracture repair.*

[1] The slot acts to hold the plate and the plate to hold the fracture. Thus there is little chance for the plate to move or the fracture to slip when the screws are applied.

[2] We now have several similar cases and all follow the same pattern. The internal immobilization has been excellent and sufficient, and the screws have not appeared to become loose or change position. Each has been characterized by early and abundant new bone. One patient, an Italian with powerful muscles, had radius and ulna plated and returned to work as a stone mason in five weeks. Later these cases will be grouped and reported.

The practice of nailing and pinning fractures of the neck of the femur has lessened the need and degree of external

FIG. 3. Eight days after operation. Patient making first efforts to exercise muscles and joints. Light calisthenic exercise only. No passive or forced motion. Only active and voluntary. Opposite arm used to assist and carry weight of gravity of injured arm. Light surface message to promote lymphatic and venous flow.

immobilization. Effective internal immobilization has resulted in more freedom, mobility and use of the leg, more frequent consolidation and more rapid recalcification. This in feeble, elderly women and in soft spongy bone supposedly not well supplied with arterial blood.

It is conceivable that under the influence of physiological activity the capacity of the process of repair to take up calcium from the circulating blood is increased, and that calcium is transformed into callus with the calcium crystals more definitely arranged according to the physical laws of stress and strain, than when physiologic activity is absent.

The callus observed after long immobilization in plaster is soft and without form. Hey-Groves has described such early callus as mushroom in character. And Delagenierre makes much of the fact that it lacks tensile strength, like fresh cement. The mineral elements in such callus do

not take on definite crystalline arrangement or design until subjected to function and use, until Wolff's law comes into play.

had confidence the plate would not bend or break, but were not confident about the screws holding. In none did the screws
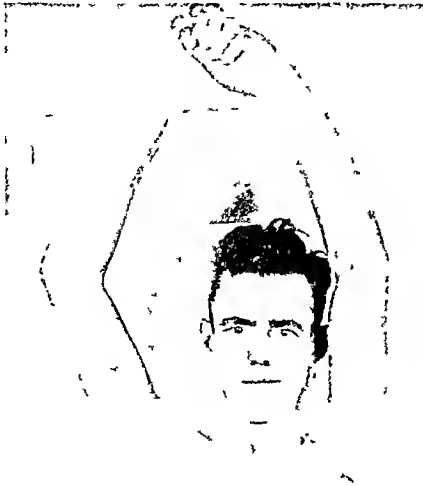
FIG. 4.

FIG. 5.

FIG 4. Two weeks after operation. Splints temporarily removed. Patient's confidence and absence of muscle inhibition used as a guide. Exercise focused on shoulder abduction, humerus rotation and elbow flexion. Arm carried in sling.

FIG. 5. Two weeks after operation. New bone shown forming around fracture.

In response to use and function, the internal structure of such callus increases in hardness, shrinks in size, and lines of graining appear.

Wolff's law is based on use and physiologic demand. It ceases to function during physiologic rest and disuse, as evidenced by the regressive changes in bone structure, reduced density and diminished mineral content, during long immobilization.

In the process of fracture repair, soluble calcium is transformed into solid crystalline calcium. This is a physical phenomenon. The process of crystallization is favored when undisturbed by motion. Agitation interferes with and slows crystallization, and changes it into an amorphous mass, like sandstone in contrast to granite.

In the earlier group of cases in which the angle plate was used, the limb was encased in plaster, much the same as after applying a flat plate of the Lane type, though the plaster was not carried quite as much above or below the site of fracture. We

appear to become loose. This gave us confidence to omit external immobilization and watch for clinical evidence of false motion and weakening of the internal fixation, study roentgenographs for change in position of plates, screws or fracture, and take note of the deposit of new bone.

It is obvious that no matter how strong a plate, its power to hold a fracture depends on how firmly the screws hold it to the bone. If the internal fixation is to be strong, the screws must *obtain and retain* a firm hold, and if the screws fit poorly or engage only the proximal cortex, the immobilization will be weak. For this reason considerable pains were taken and numerous tests made to find the type of screw and the method of its application to secure the firmest hold. To date, it has been found that long protruding screws with deep wide threads obtain the firmest hold, that the hold is greatest in the distal cortex with the butt of the screw loose in the proximal cortex. Thus far no harm has been ob-

served from protruding screw ends which appear to be soon covered with scar tissue or callus.

exceeds the pressure exerted by screws when plates are applied to the long bones.

It seems reasonable to ascribe erosion

FIG. 6. Four weeks after operation. Effort made to arouse patient's interest in progress and recovery. Frequent short exercise preferred. Noticeable increase in confidence. Splints removed at end of third week, when fracture firm clinically and roentgenographically, plate and screws unchanged and imbedded in callus.

FIG. 7. Four weeks after operation. Deposits of new bone increasing. Protruding ends of screws have proved harmless and are soon covered with scar tissue or callus. Many war veterans are carrying sharp-pointed and sharp-edged pieces of metal buried in the tissues, without apparent discomfort or harm.

Various causes have been advanced to explain why screws become loose. It is possible that in some cases screws become loosened by force, by involuntary muscle jerks within a plaster cast, and once loose they easily pull out, especially in the early stages. The better anchored the screws, the less likely for this to happen. Sir Robert Jones observed that the more effective the artificial splinting, the less the tendency for muscle splinting and muscle spasm, that this was a guide to, and proof of, the excellence of the surgical immobilization.

The writer has removed many screws in the past which were just as tight when removed as when put in. In more than 150 fractures of the patella wired with stove wire, the writer has not observed pressure atrophy in the drill holes, and pressure on the bone by the wire from direct pull of powerful muscles like the quadriceps, much

around screws to something other than pressure. It is true that pressure causes atrophy of bone. Atrophy of the vertebrae occurs from pressure of aneurysms and soft tissue tumors. But the process is slow. And screw pressure ceases with union.

Erosion of bone around buried metal plates and screws has been attributed to electrolysis, and physiologic chemists have been put to it to explain electrolysis as a cause of erosion of bone, in the absence of atrophy around plates and screws. Sherman, who has plated more than 4,000 fractures over a period of twenty-five years, has observed no harmful effects, early or late, from buried plates, and it is his practice to leave the plates in.

It is not stretching the imagination to conceive that organisms may account for bone erosion around screws. It is doubtful the degree of inoculation, the number, kind, and potency of the organisms introduced, and how many survive. Also, the

FIG. 8. Six weeks after operation. Patient returned to light work at end of fifth week. At no time was there pain or sensation at site of fracture after wound healed. Patient had confidence in strength of plated humerus from the beginning. More importance is attached at first to preserving muscle elasticity than developing muscle power, which comes later of itself.

FIG. 9. Six weeks after operation. New bone bridging line of fracture and cuff of new bone around shaft.

blood stream is not free of floating organisms. Though rare, hematogenous infections of closed fractures do occur, but they are usually of low virulence. It is not unreasonable to conceive that organisms, gaining entrance, may attack the traumatized bone, particularly the exposed cortex in drill holes, causing necrosis. Bone is mineral-bearing, mesoblastic tissue, and, according to Kahn, ranks lowest in resistance to infection. It is thus most vulnerable to organisms of low virulence and not great in number. It is possible that there are different degrees of infection after plating operations, varying from undeniable suppuration, with "laudable" pus, to a wound with an innocuous sinus which leaks for a short time, to simple isolated necrosis of bone around screws and plates. Old healed compound war fractures, if opened, are commonly found to contain locked-in organisms which, mostly anaerobic and virulent, are subject to culture. The vulnerability of bone is evidenced clinically by the severity of acute osteomyelitis and the chronicity of old infections.

if any open operation is free from inoculation, in spite of the aseptic ritual observed at the operating table. It is a matter of

CONCLUSION

The writer recalls the enthusiasm for plating fractures which followed Lane's visit to this country nearly thirty years ago. This enthusiasm changed to quite the opposite a few years later, due mainly to the prevalence of infection following open operations on fractures. Many had neglected to heed Lane's counsel and admonitions. Today bone and fracture operations are better understood and practiced.

To any one using this plate, the technique described will prove helpful. It is obvious that if the internal immobilization is to be strong, the screws must hold the plate firmly to the bone and the surgeon should take as much pains as a cabinet-maker in introducing the screws.

Removable splints which do not interfere with joint motion are used to protect the fracture during the early period of repair and are removed when clinically the immobilization appears firm and the roentgenogram show increasing deposit of new bone and the screws in place.

Should the roentgenograms reveal loose or displaced screws, or should false motion appear at the point of fracture, the natural thing would be to immobilize the limb in plaster or apply a suitable splint.

There is no reason not to expect satisfactory internal immobilization if screws get and retain a firm hold. Our experience to date is that the screws have held, none has become loose or moved, and fracture repair has begun early and progressed to early consolidation.

It is reasonable to expect that the most favorable results will be obtained when operation is performed early while the muscles are still tractable and the fracture surfaces come together accurately, when the time of operation is short, and when care is used to limit tissue handling and wound innoculation.

REFERENCE

Hawley, G. W., and Padula, R. D. A bone plate which will not bend or break. *J. Bone & Joint Surg.*, 20: 469 (April) 1936.

PRESENT STATUS OF CHEMOTHERAPY FOR INFECTIONS OF THE URINARY TRACT*

WILLIAM F. BRAASCH, M.D.

Section on Urology, The Mayo Clinic

ROCHESTER, MINNESOTA

IN considering the recent developments in chemotherapy for infections of the urinary tract I will confine myself largely to a discussion of the two compounds which have been developed recently and which are most widely used for this purpose, namely, mandelic acid and sulfanilamide. These compounds have largely displaced a host of other so-called urinary antiseptics which have been variously employed in the past decade. Medical literature of a year or two ago contained many articles concerning the therapeutic merits of mandelic acid and indications for its use. During the past year there has scarcely been a medical journal that has not contained an article concerning the action and the use of sulfanilamide. In fact, so much has been written regarding these drugs that anyone who is at all conversant with current literature has in his possession most of the investigative and clinical data concerning them.

A general survey of the literature regarding these two drugs impresses one with the fact that two entirely different principles of antisepsis are involved. Mandelic acid is an outgrowth of the use of acidification of the urine as a bactericidal agent. In fact, there are those who maintain that the greatest antiseptic action of mandelic acid lies in its ability to acidify the urine and that the specific antiseptic qualities of the drug itself are of less importance. We know that mandelic acid is not of value unless the acidity of the urine is such that the pH is 5.5 or less, and preferably 5.0. The action of sulfanilamide, on the other hand, rests on an entirely different basis. It certainly has no relation to acidification of the urine;

in fact the contrary is true. The exact method by which compounds of sulfanilamide exert their influence has not been proved. This much is true: the action of the drug in the urinary tract differs from that in the other tissues of the body. Its concentration in the tissues and in the body fluids is much less than that in the urine and if the concentration of the drug were the same in the urine as it is in the tissues it would be inadequate for the elimination of bacteria in the urinary tract.

Judging from the recent observations of Osborne and others, it is apparent that sulfanilamide does not have direct antiseptic action in the body fluids, but seems to have a toxic effect on bacterial growth and is indirectly bacteriostatic. Osterberg and Helmholz have shown that sulfanilamide is eliminated in the urinary tract in such concentration that it is bactericidal. The concentration of free sulfanilamide in the urine is 60 to 125 mg. per 100 c.c. of urine and that of conjugated sulfanilamide, 50 to 100 mg. per 100 c.c. of urine, provided that a dosage of 45 to 60 mg. of sulfanilamide is administered daily. Its concentration in the blood when given in such a dosage is usually 6 to 9 mg. per 100 c.c. of blood. Both the free and conjugated forms seem to be equally bactericidal. The bactericidal value of the drug seems to be enhanced in the presence of an alkaline urine.

ORIGIN AND DEVELOPMENT OF MANDELIC ACID AND SULFANILAMIDE

Before going any further into the merits of these two drugs, let us first review their origin and development, which reads like an international romance.

---

* Read before the Genitourinary Section of the New York Academy of Medicine, February 16, 1938.

Mandelic Acid. A brief review of the discoveries leading to the development of mandelic acid is as follows: (1) the discovery by German chemists that certain media when acidified were bactericidal to the colon bacillus; (2) the discovery by Shohl and Janney that the growth of the colon bacillus in urine could be inhibited when the urine was acidified by means of drugs given orally; (3) the discovery by Nielson that exclusion of carbohydrates from the diet caused the urine to become acidified and bactericidal; (4) the independent discovery and application of this principle by Clark and Helmholz who used a ketogenic diet with additional acidification and thereby successfully eliminated urinary infection; (5) the discovery by Fuller that beta-oxybutyric acid is the acid radical present in ketonurine and that it has bactericidal properties; and (6) the discovery by Rosenheim that mandelic acid, an exogenous acid which has a chemical composition similar to beta-oxybutyric acid is excreted intact by the kidney, and has bactericidal properties when administered in the treatment of urinary infection.

Sulfanilamide (*para-amino-benzene-sulfonamide;* H_2NO_2S⟨⟩NH_2). Even more interesting in development has been the elaboration of prontosil and sulfanilamide; its study would repay anyone who is interested in the details of its chemical development. It will be necessary for me to confine myself to a synopsis of the more important stages of its development, which have been described by Schulte:

1. The drug was first mentioned by Gelmo in 1908, while working on the chemistry of azo dyes for the German dye industry.

2. In 1913, Eisenberg discovered bactericidal powers in certain azo dyes in vitro and conceived the possibility of employing chrysoidine in therapeutics.

3. In 1917, Jacobs and Heidelberger, working at the Rockefeller Foundation, synthesized meta-amino-benzene-sulfonamide and other related products. They stated that these substances were highly bactericidal in vitro and planned to investigate this further but, unfortunately for American medicine, this never was done.

4. In 1926, Ostromyslensky introduced pyridium into American therapeutics (mallophene-serenium).

5. In 1932, Mietzsch and Klarer synthesized prontosil, prontosil rubrum, prontosil flavum, and prontosil tablets, or sulfonamido-chrysoidine, which was, however, very insoluble in water.

6. In the same year, Domagk, working with these chemists, discovered the efficacy of this and other closely related azo dyes in streptococcic septicemia of mice. One of these compounds was patented under the names of streptozon, streptozon S, prontosil 2, prontosil red, prontosil S, prontosil soluble, prontosil solution, or D-5214.

7. In 1913, Domagk and others reported its use in the treatment of streptococcic infection.

8. The Trefouëls, Nitti, and Bovet, working in Fournot's laboratory at the Pasteur Institute, recalled the hypothesis advanced by Heidelberger and Jacobs that sulfonamido-chrysoidine, when broken down, formed para-amino-benzene-sulfonamido, which we call "sulfanilamide." They assumed that this was the effective portion of the prontosil molecule and they were the first to show its bactericidal power.

9. English chemists, Fuller, Colebrook, Buttle and O'Meara, showed that prontosil could be reduced to sulfanilamide.

10. It was in the English laboratories that Long and Bliss were informed of these new chemotherapeutic agents and they took these ideas back to America. They proved that sulfanilamide is formed in vitro by reduction and they found that cysteine was an ideal reducing agent for their experiments. They found that sulfanilamide was bacteriostatic in vitro, whereas prontosil and cysteine were not.

11. Marshall, Cutting and Emerson found that part of the sulfanilamide ingested is excreted in the urine without

chemical change. A small part is converted to para-acetyl-amino-sulfonamide.

12. Rosenthal synthesized di-sulfanilamide but its disadvantage was that it was only very slightly soluble in water.

TREATMENT

In a consideration of the respective therapeutic merits of these two drugs, we find that both have their places in the treatment of urinary infection. The choice depends largely on such indications as idiosyncrasy, type of bacteria present, the patient's general condition, the age of the patient and coincident complications. As a general rule, it may be said that therapy with mandelic acid causes less reaction and is usually not so toxic as sulfanilamide. On the other hand, among the advantages of sulfanilamide may be included a more rapid and spectacular elimination of bacteria, a wider range of types of bacteria affected, and an independence from the limitations of the process of acidification.

There is considerable difference of opinion as to the desirability of the two drugs. Some urologists, particularly if they have treated a series of patients who had severe reactions following the use of sulfanilamide, use mandelic acid in most cases of bacilluria, employing sulfanilamide only when the administration of mandelic acid fails to have the desired effect. Bacilluria in the female urinary tract usually can be controlled easily by the administration of mandelic acid and with a minimum of reaction. On the other hand, there are those who are impressed by the ease with which bacteria frequently are eliminated by the administration of sulfanilamide and they use this drug routinely. In men, because the infection usually has its origin in the prostate gland and seminal vesicles, it is generally advisable to use sulfanilamide on account of its bacteriostatic action in these foci. There are definite indications for the use of both drugs and they should be used interchangeably.

The administration of mandelic acid is likely to cause acidosis among old adults.

Acidosis is also associated with the administration of sulfanilamide, although less frequently. We usually give mandelic acid in an effervescent form of a flavored elixir of ammonium mandelate (20 per cent), which is usually better borne than the more concentrated solutions. In a few cases, a 10 per cent aqueous solution of the ammonium salt is best taken. More recently the use of a calcium preparation in tablet form, in the hands of some, has been associated with less reaction than the liquid form. If one type of solution is not tolerated well, it is advisable to try another. Although the majority of patients do not have a strong dislike for the various forms of mandelic acid, some will have a violent distaste for it in any of its different pharmaceutical forms. If such occurs, it will be necessary to use sulfanilamide in the place of mandelic acid. Occasionally, placing the intolerant patient at rest in bed seems to lessen such reactions.

Although Helmholz and Osterberg have shown that the administration of mandelic acid to dogs is followed by an increase in the concentration of blood urea, this is seldom observed among human beings, provided that renal function is normal. When renal function is impaired, the administration of either drug may further reduce renal activity and cause various symptoms of toxemia. Although in most cases either drug will cause toxic reactions to a varying degree, as a rule sulfanilamide is the better borne of the two, because acidosis frequently follows the administration of mandelic acid. In many cases of renal dysfunction neither of the drugs is eliminated in sufficient concentration in the urinary tract to be of much value. In cases in which the reduction in function is the result of long-standing urinary obstruction sulfanilamide is better tolerated than mandelic acid and is more efficacious in the treatment of such a condition than it is in cases of glomerular disease. The administration of these drugs is often followed by a great decrease in renal function, as is shown

by a rapid increase in the concentration of blood urea.

There is some difference of opinion as to the efficacy of sulfanilamide as a preoperative and postoperative aid. It has been our experience that in the presence of severe infection in the urinary tract sulfanilamide can sometimes be employed to advantage in diminishing the degree of infection prior to operation. As a postoperative aid, however, it has not proved to be of much value in our hands. Thompson employed it after prostatic resection in a series of cases in which a variable degree of postoperative urinary infection was present. He failed to see that it made any difference in the degree or duration of the postoperative infection and he has discontinued its use. It would seem that in common with all forms of chemotherapy, this drug is not efficacious in the presence of tissue which has recently been operated on. It may be of value, however, in the treatment of infection that persists some three or four weeks after operation when the tissues have healed.

The drug proved to be of comparatively little value in the presence of stone, tumor, or lack of drainage in any portion of the urinary tract. It is seldom that much benefit results from chemotherapy in the presence of drainage tubes in either the upper or lower portion of the urinary tract.

DOSAGE

The dosage of mandelic acid is more uniform than that of sulfanilamide. It is usually necessary to use 10 to 12 Gm. daily to secure results. The dosage of sulfanilamide, however, varies widely with the age and general condition of the patient and with the type of infection. The administration of an initial large dosage is not so essential in the treatment of infection in the urinary tract as it is in the treatment of a fulminating type of general infection and there is a tendency toward administering a lower dosage than was at first thought necessary. The dosage which is tolerated by

the young adult is decidedly different from that tolerated by the older adult. The majority of young adults experience comparatively little reaction from an initial dosage of 60 to 80 gr. (4 to 5.2 Gm.) and after a few days it can usually be reduced to half that dosage. Among adults aged 40 years or older, and among many younger women, a more cautious approach is desirable. Severe reactions with collapse are not infrequent among patients more than 60 years of age. Idiosyncrasy on the part of the patient appears when least expected and must always be anticipated.

Problems associated with the use of sulfanilamide in the treatment of widespread infection in the tissues are entirely different from those encountered in the treatment of infection confined to the urinary tract. Although the concentration of sulfanilamide in the tissues sufficient for bacteriostatic action is much less than that required for a like purpose in the urinary tract, the administration of a very heavy dosage is frequently necessary in order to have any effect on systemic infection. Cases are sometimes encountered in which it is necessary to give as much as 120 gr. (8 Gm.) daily, and if improvement is not noted, it has been advisable to give in addition, subcutaneous injections of prontosil. Prontosil apparently, in some of these cases, not only acts differently from sulfanilamide, but also seems to add to the efficacy of sulfanilamide.

EFFECT ON RENAL INFECTIONS

Failure in eliminating renal infection may be owing to widespread cicatricial changes in the renal tissue and retarded renal drainage resulting from chronic infection. Nevertheless, I have recently observed several cases of chronic pyelonephritis of this type in which the condition improved after the administration of repeated courses of sulfanilamide. I recall one case of cicatricial chronic pyelonephritis in particular, which had existed for twelve years, with frequent periods of

hematuria, and which failed to respond to any form of treatment, local or general. The infected urine gradually cleared and finally, after repeated courses of sulfanilamide, became crystal clear and sterile on culture. If the course of treatment is repeated at short intervals in some cases, the infection is removed. This procedure should be tried in stubborn cases. Sometimes, disappearance of the bacteria does not occur until one or two weeks after administration of the drug has been discontinued.

A working knowledge of bacteriology is necessary for the intelligent use of chemotherapy. The administration of mandelic acid will usually control the ordinary forms of bacillary infection. When persistent types of bacilli are present and those which have urea-splitting properties, such as proteus, sulfanilamide is to be preferred. Although mandelic acid has but little effect on the various coccal infections among adults, it has a specific action on Streptococcus faecalis, against which sulfanilamide has little or no inhibiting effect.

Sulfanilamide is usually of greater value when bacteria are found in a persistently alkaline urine.

We still must frequently fall back on the use of arsenical preparations, such as neoarsphenamine. This is particularly true with regard to coccal infections of long standing, and in cases in which cocci occur in a urine which has a low hydrogen ion concentration. It has been our experience, however, that neoarsphenamine is only efficacious in eliminating coccal infection in approximately 50 per cent of uncomplicated cases. By uncomplicated cases I mean those in which complications such as stone, obstructed urinary drainage, or chronic cicatricial changes in the renal tissue are not present. Because the presence of stones in the kidney is so often accompanied by coccal infection, the elimination of such infection is often a baffling problem. It is evident that the ideal disinfectant for coccal infection still remains to be discovered.

COMPLICATIONS

A special problem presents itself when several types of bacteria are present in the infected urine. In a recent review of 200 cases of chronic pyelonephritis, I found that mixed infection was present in more than 10 per cent of cases. I believe this percentage is greater when the subacute type of infection exists. The combination which is probably most frequently observed is that of the colon bacillus with Streptococcus faecalis. The coincidence of the colon bacillus and Aerobacter aerogenes, and the colon bacillus with various types of cocci occurs often. In fact, all sorts of combinations of bacteria have been encountered. If a course of mandelic acid is given and if the urine fails to become clear, coccal infection frequently will be found to persist. If this infection does not disappear with the use of sulfanilamide, neoarsphenamine should be administered. If, after employing sulfanilamide as a primary therapeutic agent, bacilli are eliminated and Streptococcus faecalis persists in the urine, the organism will usually be eliminated subsequently by the giving of a few doses of mandelic acid together with supplementary acidification of the urine.

When infection persists in spite of all the various forms of therapy, it must be inferred that there is some cause for the persistence of the infection. That cause must first be eradicated. In order to ascertain the results obtained with chemotherapy, the cases should therefore be divided into groups, one group consisting of complicated cases and the other of uncomplicated cases.

The most frequent complication which hinders elimination of bacteria among men is prostatic infection. Bacteria are often eliminated from the bladder, only to return from a persistent prostatic infection. It is, of course, necessary that vigorous treatment should be directed to this region. As has been emphasized previously, prostatic infection may be secondary to other foci of infection and such primary foci should

first be eliminated. Particularly is this true of tonsillar infection. Sulfanilamide has been found to be very helpful in the treatment of chronic prostatitis and its administration should be repeated at intermittent periods. Prostatic massage should be employed at the same time and experience has shown that administration of sulfanilamide facilitates the results of massage to a great extent. It has been our experience that reduction in the degree of prostatitis can be brought about in at least two-thirds of the cases. A great reduction in the time required by massage for the elimination of infection is observed. Incidentally, relief of associated nonspecific discharge also occurs. Sulfanilamide is probably excreted in the prostatic secretion in insufficient concentration to be bactericidal. The exact manner by which the prostatic infection is influenced is not known, but apparently, sulfanilamide exerts the same toxic influence on bacteria in the prostatic tissues that it does on bacteria in other tissues of the body.

It is of interest to review some of the complications which are sometimes observed following the use of sulfanilamide: (1) Nausea occurs to a slight degree in most cases but is usually overcome by reducing the dose for a day or two. (2) Headache and dizziness, although frequently present, are not usually severe enough to incapacitate the patient. Such symptoms can usually be overcome by a temporary reduction of dosage or by cessation of administration of the drug. Either of these complications may occur on the first trial of the drug but disappear subsequently. (3) Skin eruptions such as urticaria or a "contact" eruption may be limited to the surfaces of the skin exposed to sunlight or may involve the skin of the entire body. Such eruptions always disappear promptly after the administration of the drug is discontinued. (4) Cyanosis may occur and may be due to hemoglobinemia, sulphemoglobinemia, or methemoglobinemia. It disappears on discontinuing administration of the drug and is of no

great clinical significance. (5) Fever of variable degree may occur either early or late in the course of administration of the drug. In some cases, it has been observed within twelve hours after the administration was begun. Although usually ceasing within a day or two after administration of the drug is discontinued, it may persist longer. (6) Acidosis may occur as previously mentioned, and is a signal for immediate discontinuance of use of the drug. The symptoms may continue several days afterward. (7) Leucopenia, usually controlled by transfusions of blood, may persist in spite of various methods of treatment employed. The chemical formulas of the sulfonamide group of drugs are based on the benzene ring. It should be remembered that benzol, which belongs to this group, has been employed to reduce the formation of leucocytes in cases of leucemia. This same action may explain the appearance of leucopenia and agranulocytosis following the use of sulfanilamide, as this drug also is a compound containing the benzene ring. The advisability of using a substance which is definitely toxic may be questioned. It must be remembered, however, that according to the science of pharmacology small doses may be of great therapeutic value, whereas large doses may act as poisons. It has been shown experimentally that a low concentration of sulfanilamide in the blood and tissues usually does not affect the production of leucocytes, although when the drug is used in too large or too long continued doses the number of leucocytes may be diminished. It seems that there are certain patients who have an allergic reaction to the drug the nature of which seems to be a mechanism of profound interference with the production of leucocytes. (8) Only occasional cases in which death has occurred have been reported and the cause in some cases has not been known. In one of our cases, death followed the performance of transurethral resection for carcinoma of the prostate gland. Sulfanilamide was administered for a week without any untoward

result; then fever suddenly developed and the patient died a week later; a fever persisted until death. At necropsy a definite cause of death could not be determined.

Although sulfanilamide represents a tremendous advance in elimination of infection, nevertheless the ideal bactericide has not yet been found. The toxic manifestations of this drug are a serious handicap to its employment in many cases. A compound, distinctly less toxic and supposed to replace sulfanilamide, was discovered recently by Domagk and his co-workers. Its formula is as follows:

$$H_3C \diagdown$$
$$\quad\quad NO_2S\langle\quad\rangle HNO_2S\langle\quad\rangle NH_2$$
$$H_3C \diagup$$
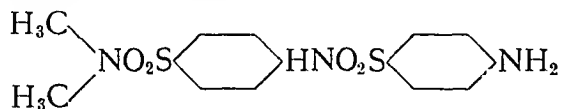
Grütz found this to be very effective against staphylococci, colon bacilli, anaerobic gas bacilli and the gonococci. Chemically it is di-methyl-di-sulfanilamidc. It was referred to tentatively as D-373 and was also known as uliren. In this country it was called diseptal. It was found that this drug was distinctly less toxic than sulfanilamide, and many patients who had a severe reaction following the use of sulfamilamide had few, if any, symptoms as the result of its administration. This drug also was supposed to be somewhat more bacteriostatic than sulfanilamidc. Owing to the diminished toxic reaction di-methyl-di-sulfanilamide could be employed longer and in larger doses than sulfanilamide.

This drug was employed at The Mayo Clinic in a considerable number of cases with excellent results. Everything went well until two patients to whom it was given over a period of two or three weeks suddenly experienced wrist-drop and manifested other evidence of toxic neuritis. Other observers in this country have had similar experiences, although in Germany such complications have not been reported. It was found that such reactions took place only in cases in which large doses were continued over a period of several weeks. It has been the experience of others, however, that this compound can cause serious nerve injury. The drug was taken off the market at once by the manufacturers and efforts are being made to eliminate its toxic qualities.

Similar compounds which will not cause systemic toxemia and will be even more bactericidal than sulfanilamide or di-methyl-di-sulfanilamide, will undoubtedly soon be made available. The future of chemotherapy in combating infection would seem to offer great possibilities.

TRANSPLANTATION OF THE URETERS*

ELMER HESS, M.D., F.A.C.S.

Chief, Urological Department, St. Vincent's Hospital; Senior Urologist, Hamot Hospital

ERIE, PENNSYLVANIA

INTRODUCTION

FOR years it has been a recognized fact that there were a few pathologic conditions that required for their relief the short-circuiting of the urine. Coffey was the modern pioneer in this work, and it was due to his experimental and clinical efforts that the various transplantation procedures of today were devised. That any operation of this nature has a high mortality and morbidity goes without saying and it usually is indicated only in those cases where death is preferable to the continued subjective suffering of the unfortunate individual. The procedure, then, is never one that will become common practice in the hands of any one man and as the indications are rare very few men will ever acquire sufficient experience to become real experts. However, many competent urologists will do a few of these operations in a lifetime of specialized effort. To those of us who have had even a small number of these cases, falls the often unpleasant task of reporting our experiences. If this is done frankly, only good can come from open discussion of our successes and, more particularly, our failures. This is true if we have sufficient interest to follow our failures to the autopsy table when the reason for them is often very plainly written.

The work of Coffey, the Mayos, Beer, Mathé, Kirwin, Counsellor, Hinman, and many others is now familiar. This report, therefore, will be based solely on personal experiences.

INDICATIONS FOR VARIOUS TYPES OF URETERAL TRANSPLANTATION

1. Carcinoma of the bladder.
   (a) Without ureteral obstruction.
   (b) With ureteral obstruction, unilateral or bilateral.
2. Carcinoma of the cervix (postradiation). With ureteral obstruction, unilateral or bilateral.
3. Vesicovaginal fistula.
4. Interstitial cystitis, non-tuberculous.
5. Interstitial cystitis (healed contracted tuberculous bladder).
6. Anomalies of the lower urinary tract —epispadias and extrophies where there is complete lack of urinary control.

There have been many types of transplantation procedures designed to aid these various conditions. Where the ureters, one or both, have been obstructed low in the pelvis, nephrostomy with permanent drainage has been done in a few cases. Usually this has been only a temporary procedure to save the kidney until the obstruction could be relieved. I have never been forced as yet to do a permanent nephrostomy.

Occasionally, due to stricture of a ureter either from intrinsic or extrinsic causes, it is necessary to transplant it into the bladder. This is not a particularly difficult problem and is usually successful. I have done this operation once with a good result. Occasionally, it should be good treatment to do an anastomosis between the ureter and the bladder or even between the pelvis of the kidney and the bladder. This latter procedure was done by me for the first time in April, 1928, and is the only case of its kind on record. The indication was a huge hydronephrosis of the right pelvis of a horseshoe kidney due to low obstruction of the ureter. The operation was a perfect success and the patient is still living and in good health.

Transplantation of the ureters to the surface of the body has been done frequently by Beer, Mathé, and others. The indication is usually carcinoma of the bladder. The operation is intended to short-circuit the urine away from the bladder so that this organ may be totally destroyed or removed and the carcinoma thereby cured. Openings of the ureters are then treated by placing indwelling catheters in them and the urine is carried to containers strapped to the legs. This operation, while not without risk, is an extraperitoneal procedure and relatively simple. The question of morbidity immediately arises. I have never done this operation by choice. I know that Beer and others follow this procedure, reporting quite a number of patients who are living and enjoying life.

My own experience with the operation has been limited to ten cases where the lower end of both ureters had become occluded as a result of radiation therapy directed at a cervical carcinoma. These were virtually emergency operations to relieve the obstructive hydronephrosis. Most of the patients died in a few months; two lived a year, and all died of urosepsis. I have never deliberately done the operation for carcinoma of the bladder, believing that if any transplantation procedure were done it would be better to transplant into the bowel rather than to the skin. Because of the morbidity, I cannot see the reason for transplantation to the skin for carcinoma of the bladder even though there is little mortality. If we transplant ureters into the bowel for epispadias and exstrophy, why create this condition? I am willing to admit that the operation is occasionally justified because at times life is greatly prolonged and some people will prefer a more or less miserable life to death.

This statement brings up an important problem in the handling of all of these cases. In the case of a child, the parents should be told of the high mortality and morbidity. In an adult the person upon whom any of these operations is to be performed should be taken into the physician's confidence and none of these surgical procedures should be performed unless *elected* by the patient. Many will refuse such procedures when they fully realize the situation even if they recover.

TRANSPLANTATION OF URETERS INTO THE BOWEL

The first indication for transplantation of the ureters is cancer of the bladder.

Case 1. C. J. D., male, age 61, was admitted on August 5, 1930, with a diagnosis of carcinoma of the bladder. The patient's chief complaint was blood in the urine, with occasional inability to void. He had some frequency, urgency, and nocturia. He had been perfectly well until two and a half years before, when he noticed that his urine was blood-tinged and that the condition was getting worse.

It was impossible to see the entire tumor through the cystoscope. There was a large carcinoma at the base of the bladder, involving the trigone and the bladder itself.

Laboratory Report. Red blood cells numbered 4,520,000; white blood cells 8,000; hemoglobin 70 per cent; blood urea nitrogen 12.6; blood sugar 150; Wassermann negative. Culture of the urine showed many colonies of staphylococci, no tubercle bacilli, but a large number of pus cells. The tumor tissue from the bladder showed carcinoma of the urinary bladder with evidence of rapid growth. Microscopic section showed typical carcinoma of the bladder epithelium, many hyperchromatic nuclei and large numbers of mitotic figures. The tumor was papillary in structure in some places and showed mild inflammatory reaction.

On August 6, bilateral transplantation of the ureters was carried out using the Coffey catheter technique. On August 7, the lungs were clear. The abdominal peristalsis was exaggerated. On August 8, the patient complained of pain in the abdomen. An enema was ineffectual in relieving the abdominal distention. On August 10, the patient died due to obstruction of the bowel.

Autopsy. Incision in the abdomen showed no excess of peritoneal fluid and there was no evidence of peritonitis. The muscles were deep red in color. The lower bowel from the cecum to the splenic flexure and small bowel was filled with fluid and greatly distended with

gas. There was no evidence of adhesions, except as noted later, and no evidence of loss of peritoneal or mesentery fat. The appendix was in the right position, long but normal. Dense adhesions bound the bowel for a length of about 4 inches at the splenic flexure, the adhesions contracting the descending colon to such an extent as to give complete obstruction at this point. From there on the descending colon collapsed.

The anastomosis of the two ureters was completely healed over with some few inflammatory adhesions at the site of the anastomosis, but the condition over the sigmoid at this site showed rapid healing. There was no other inflammatory evidence except that due to the trauma of operation. The ureters, where they had been severed at the bladder, were closed and there was no leakage from the bladder.

The bladder was small and empty and was removed with the prostate. The prostatic urethra and base were filled with mushroom growth, carcinoma. The bladder walls were markedly hypertrophied.

The liver was free from metastasis, and the kidneys were normal in size. There was no enlargement of any of the vertebral glands. There was no excess of pleural fluid on either side and no adhesions in either lung except for a few posteriorly to the parietal pleura on the left side. The autopsy was otherwise negative.

Microscopic sections of kidney revealed no inflammatory reaction. All sections were essentially normal renal structure and there was no inflammatory reaction in the pelvis of either kidney.

Pathologic Diagnosis. Death caused by obstruction of the bowel. No evidence of peritonitis.

Case ii. W. R., male, age 64, was admitted on September 26, 1930, with a diagnosis of carcinoma of the bladder. The patient's chief complaint was blood in the urine, which he noticed on September 20. He had no other discomfort at this time. He had lost no weight.

On September 23, cystoscopy revealed a carcinomatous growth on the left lateral wall of the bladder, which was so large that it could not be entirely visualized with the cystoscope. The laboratory report stated that a section of tumor tissue showed typical carcinoma of the bladder epithelium with mitotic figures. The blood chemistry was normal; Wassermann and

Kahn tests negative; blood counts were all within normal limits.

On September 28, the patient was operated upon. Both ureters were transplanted into the rectum, using the Coffey catheter method. On September 29, urine dripped freely from the catheters. On September 30, the bowels moved slightly, urine dripped freely. On September 31, the patient was unable to move the bowels, but urine dripped freely. The patient died suddenly from cardiac dilatation.

Autopsy. The left side of the chest showed a marked prominence over the cardiac area. The abdomen was moderately distended. Primary incision revealed a moderate quantity of bright yellow adipose tissue. The muscles were of good color. The coils of the small intestine were found to be greatly congested; careful observation revealed here and there a small quantity of exudate, which joined adjacent loops of the intestine together. Upon pushing the coils of the intestine aside, it was observed that there was a slight excess of peritoneal fluid, perhaps 15 or 20 c.c., markedly turbid and somewhat blood-stained. The omentum showed little exudate and the anterior surface of the stomach was only slightly congested. There appeared to be no definite exudate upon the lower surface of the diaphragm nor the external surfaces of the spleen or liver.

Ureteral catheters extended up both uretres nearly to the kidney pelvis and down through the rectum, leaving the body at the anus. No definite seep from the rectum into the peritoneum had occurred. (Fig. 5.) However, on the right side at or near the point of entrance of the ureter into the rectum there was a considerable quantity of exudate, which was more abundant in this region than elsewhere. Careful examination revealed no other source of the peritoneal exudate.

Culture of the peritoneum showed several colonies of staphylococci among which were seen small Gram-positive diplococci and small chains of three and four cocci (Gram-positive). The pericardial cavity contained about 20 c.c. of clear, straw-colored fluid. There were no pericardial adhesions and both layers of pericardium were smooth and glistening. The only pleural adhesion was a slight one between the space of the left lower lobe and the diaphragm. There was no excess of pleural fluid and both layers of pleural sacs were everywhere smooth and glistening. The pulmonary artery con-

tained fluid, fluid blood, and post-mortem clots. Smears of peritoneum showed a moderate number of polymorphonuclears, large mononuclear wandering cells, and occasional small Gram-positive diplococci. The heart was greatly enlarged, showing dilatation with hypertrophy.

Microscopic examination revealed a mild mechanical pyelitis in the right kidney due to mercurochrome, but this did not extend into the renal substance.

Pathologic Diagnosis. Ileus; mild peritonitis (this might have cleared up) and acute dilatation of the heart.

CASE III. M. Z., male, age 51, was admitted to the hospital on March 14, 1931 with a diagnosis of vesical calculus. The patient's chief complaints were suprapubic pain, frequency, incontinence and hematuria, all of which had been present only three weeks. Cystoscopic examination revealed a large tumor involving most of the bladder wall; this proved to be a squamous cell carcinoma. The blood pressure was 120/80. There had been a previous lower abdominal operation.

On March 16, both ureters were transplanted. The sigmoid was found to be bound down in the right pelvis by marked adhesions, and was freed with difficulty. Both ureters were badly dilated. They were transplanted by the Coffey catheter technique. An average of 24 ounces of urine was drained every twenty-four hours. On the fifth day the catheters were removed and convalescence was deemed satisfactory. On March 29, thirteen days after operation, the patient became distended and all attempts to relieve the distention failed. Death from ileus followed on March 31, fifteen days after operation.

Autopsy. The bladder was 8 cm. in length and ureteral stumps which measured about 4 cm. were attached to it. The bladder wall was greatly thickened and its mucosa soft and necrotic in appearance. Extending over the greater portion of the bladder surface was a broad area of new growth, the surface of which was like a cauliflower in appearance, while the underlying tissue was soft and friable. This malignant condition extended up over the trigone to the urethral orifice and, except for a small area on the anterior wall, appeared to have infiltrated the entire mucosa. Sections of the wall showed the new growth to have infiltrated in some places through to the peritoneal coat. No apparent metastases out-side the bladder were demonstrated. There was intense dilatation of the ileum.

The sigmoid colon extended for about 10 cm. in the right iliac fossa. The ureteral transplants were made in a diagonal direction through the intestinal wall. One of these was still intact, the other was apparently torn away by the trauma of removal. The pathway of incision through the intestinal wall and the opening through the mucosa appeared in fair condition, although infection around both of these areas was present and beginning peritonitis in the region of the sigmoid was noted. Death was due to ileus (slight beginning peritonitis).

CASE IV. A. H., male, age 35, was admitted June 2, 1937 with a diagnosis of squamous cell carcinoma. He was discharged on November 6, 1937 and readmitted on November 19. On the first admission the chief complaint was frequency and burning on urination. Cystoscopic examination revealed an extensive growth of the bladder. The first pyelograms were normal and showed normal ureters. There was no evidence of metastasis. Transplantation of the ureters with total cystectomy was planned but not done. Deep x-ray therapy failed to give any relief and the patient died of urosepsis on January 10, 1938.

Autopsy. This was entirely negative except for the following findings:

The right kidney weighed approximately 160 Gm., and measured 11 × 6 × 4 cm. The capsule was moderately adherent, the kidney pelvis much distended, containing about 30 c.c. of thick purulent-appearing fluid. In part of the kidneys, the pyramids and cortex showed much atrophy. Elsewhere the cortex was 6 to 8 mm. in thickness. No definite abscesses were noted in the kidney substance. The right ureter was much distended. It varied from about 1.5 to about 2.5 mm. in diameter and contained much thick, purulent-appearing fluid. The mucosa showed much desquamated epithelium. A probe introduced into the right ureter did not pass into the urinary bladder.

The left kidney was considerably larger than the right, weighing approximately estimated weight 175 Gm. The capsule was thin and stripped easily. The kidney pelvis was slightly distended with about 20 c.c. of thick purulent fluid.

The kidney tissue showed but little atrophy. The cortex varied from 6 to 8 mm. in thickness. In places small yellowish streaks which might

indicate diffuse suppurative nephritis, were seen parallel to the collecting tubules. No definite abscesses were seen in the kidney tissue. The left ureter, 1 to 2 cm. in diameter, was distended with purulent fluid. A probe introduced into the lower part of the ureter passed into the urinary bladder with but little resistance.

The urinary bladder was considerably enlarged, having an average diameter of about 9 cm., with walls from 6 to 10 mm. in thickness. It contained about 150 c.c. of turbid yellowish-gray fluid. In the lower half of the bladder the mucosa showed a necrotic, moth-eaten surface, with small, poorly defined elevations in some places. Apparently the whole of the lower half of the bladder was involved in an extensive malignant tumor growth, which affected the whole of the trigone and much tissue surrounding both ureters. In places the mucosa appeared as a sloughing necrotic mass. The necrosis and thickening of the mucosa extended into the membranous urethra for a distance of about 2 cm. Apparently the tumor tissue extended into the mucosa of the urethra, but the anterior and upper part of the mucosa of the bladder, while showing moderate trabeculation, swelling and edema, was apparently free from tumor growth.

Examination of the prostate gland showed no hypertrophy nor invasion by the tumor tissue. The seminal vesicles were not involved. Examination of the urethra showed no stricture or obstruction.

Microscopic. Sections of the urinary bladder showed a very widespread, far advanced, squamous cell carcinoma, tumor cells invading the bladder wall quite extensively. Numerous epithelial pearls were observed and the nuclei were large and hyperchromatic. Moderate numbers of mitotic figures were present. The tumor showed necrosis on the surface, but the deeper lying cells showed little or no necrosis. A widespread invasion of the tissue showed that the tumor was highly malignant.

Anatomic Diagnosis. Far advanced squamous cell carcinoma of the urinary bladder with extension to the mucosa of the posterior urethra and obstruction of both ureters and bilateral pyonephrosis.

This last case brings up an important point. This man had been under our observation for about six months, and ureteral implantation into the bowel with total cystectomy had been contemplated early. His original study revealed no dilatation of the ureters or pelvis. His cystoscopic study revealed such a complete involvement of the entire bladder that it seemed impossible completely to remove the disease. The operation for that reason was not done.

Autopsy revealed that death was due to urosepsis with wide dilatation of the ureters and pelvis. Strangely enough, the carcinoma (squamous cell) was completely limited to the bladder without extension or metastasis. A perfect case for transplantation and cystectomy.

All of these cases, it is noted, terminated fatally.

I am convinced from my own personal experience, small though it be, and from conversations with many men more experienced than I, that carcinoma of the urinary bladder should not be treated by ureteral transplantation and total cystectomy. However, I will not seriously criticize those who differ with me, but will present the argument from my own experiences with this entity. Three cases of transplantation are reported here—with three deaths.

In a series of fifty-seven consecutive cancers of the bladder since 1930, we have lost twenty-two patients. Several in the group of thirty-five living have had recurrences. All of these patients have been treated by the use of radium and x-ray, almost all of them without any other operative procedure. In those who died the only operative procedure was a suprapubic cystotomy. Of those who are living two have had cystotomies. One must note that a number of the dead succumbed to conditions other than their cancer; on the other hand, all the living may not, of course, be considered as cured.

Cancer of the bladder, to be cured, must be found early and treated promptly. Usually the curable types, early diagnosed, can be treated cystoscopically with radium and x-ray. If we are fortunate enough to get the cases early, before the infiltration has

involved pericystic tissue or metastasis has taken place, the vast majority can be cured. Cancer of the bladder fundus (one of its rarest locations) is as amenable to bladder resection as is cancer of the fundus of the uterus to supravaginal hysterectomy and the prognosis is usually good, particularly if followed by x-ray therapy. (This is denied, however, by some men.)

These early carcinomas may also be cured by transplantation of the ureters and total cystectomy. Both operations are so formidable from the mortality and morbidity aspects that they are absolutely unjustifiable when such excellent results can be obtained by radium, x-ray, and at the worst, simple resection or cystotomy, combined with x-ray and radium. There is virtually no mortality associated with these procedures, the morbidity is moderate, and very little time is lost.

This holds true whether the ureters are transplanted to the skin or to the bowel, although, of course, transplantation to the skin is obviously a more simple operation than transplantation into the bowel.

Cancer of the bladder that has progressed beyond the so-called early curable stage cannot be cured by these formidable procedures if it cannot be cured by the other agencies (radium and x-ray). Occasionally an apparently inoperable, incurable case of cancer will respond to these agencies, but leave the lower ureter or ureters obstructed and the bladder so contracted that the urine must be short-circuited. Then transplantation must be considered, not as a treatment of the cancer, but for the same reason that it is considered in the postoperative contracted bladder of tuberculosis. If one kidney is obstructed and its fellow normal, nephrectomy may be the elected procedure. When life is limited even if the patient survives, and when the same, if not better, results can be obtained by x-ray and radium without any drastic procedure, why subject a patient with an inoperable carcinoma of the bladder to transplantation plus total cystectomy, unless it is with euthanasia in mind?

I make this statement in spite of the fact that Beer reports excellent results from transplantation of the ureters to the skin with total cystectomy, and that Higgins advocates transplantation of the ureters into the bowel with total cystectomy in extensive carcinoma of the bladder, saying: "During the past three years, patients whose condition would, in the past, have been considered hopeless and for whom our entire reliance would have been placed on roentgenotherapy, have been given the benefit of cystectomy and transplantation of the ureters with outstanding results in some cases. Cystectomy and transplantation has now been performed in thirty-four cases of carcinoma of the bladder in our series."

In a more recent article, however, reporting this same series of cases, Higgins takes a different stand: "Less radical operative procedures should be employed in advanced cases of carcinoma of the bladder associated with large dilated ureters, pyelonephritis, and impaired renal function. Such vesical carcinomas lie beyond the range of operability for this type of surgical procedure. ... . The question is raised as to the advisability of earlier transplantation of the ureters and cystectomy before irreparable damage to the kidneys has developed."

No one, I believe, could take any exception to Higgins' indications for the operation in early carcinoma of the bladder. However, he does not give his figures concerning either mortality or morbidity.

Yet in spite of the reports of these two men, I still feel that when cancer of the bladder is diagnosed sufficiently early to be curable, transplantation of the ureters with total cystectomy is never indicated.

EPISPADIAS AND EXSTROPHY
OF THE BLADDER

When epispadias and exstrophy of the bladder exist, the indication for bilateral transplantation of the ureters into the

bowel is not questioned. I have seen four cases of this condition. In the first instance, the patient was a man in his forties, who, after the situation and risks were fully explained, refused operation. In the second case, the patient was a child of 8 weeks who had a successful transplantation of the right ureter from which he recovered; however, a month later when the left ureter was transplanted, he died of acute obstructive suppurative pyelonephritis. In the third instance, after explaining the situation to the child's mother and obtaining her permission, I treated the baby for twenty-one months to give it the best possible chance for recovery and then operated. Unfortunately, the child succumbed from the operative procedure. The last case, a boy of 13, was admitted to the hospital with the bladder closed but the penile condition not corrected. The boy was a social outcast and his teachers would not have him in school. I tried to correct the defect in the penis and bladder neck, unsuccessfully, due to lack of coöperation on the part of the boy. The parents then consented to a transplantation. This patient lived.

Case v. R. M., age 8 weeks, was admitted on October 10, 1930, for correction of epispadias with partial exstrophy of the bladder.

On October 27, an unsuccessful attempt was made to repair the exstrophy and epispadias by the Young technique. On November 13, the right ureter was transplanted by a modified Coffey-Mayo technique. The child ran a septic temperature for four or five days and urine drained from the rectum. The temperature then subsided to normal and the child voided from the rectum four or five times a day.

On December 11, the left ureter was transplanted, and again the child ran a septic temperature. He died on December 23 of acute pyelonephritis.

Autopsy. The autopsy was negative except for the following findings: The ureter transplant on the right side was healed with normal ureter and kidney. The left ureter was healed with no leakage, but the last suture bringing the layers of the bowel together had been placed too tightly, completely obstructing the ureter at

this point. The kidney on this side showed marked inflammatory and destructive lesions in the upper pole with much inflammatory reaction and minute abscesses scattered through the parenchyma. Microscopic examination showed a normal, non-inflamed right kidney. Death had been due to left obstructive ureteropyelonephritis.

Case vi. B. B., female, age 5 weeks, was admitted to the hospital on May 10, 1935. The chief complaint was irritability and excessive crying as the result of a congenital deformity—exstrophy of the urinary bladder. Nothing else was significant in the past history.

The mother and father were 19 and 22 respectively, and in good health. There was one other child, a boy, aged 2, alive and well. No history of any familial disease could be obtained.

The infant was poorly nourished, and showed no evidence of the umbilicus—a small hernia at the upper end of the bladder probably represented it. The posterior wall of the bladder was present in the lower abdomen; the mucosa appeared reddened and the ureters were draining. The urethral opening could be seen at the apex of the trigone. The external genitalia were rudimentary, pads of fat representing the labia. Another deformity was found on x-ray examination, a congenital absence of both pubic bones and their rami. The spine was normal.

Seven days after admission, the child developed a generalized rash which looked very much like measles. About eight days later, a high septic-type of temperature developed, which rose to 105 degrees; this receded on the third day. Bronchopneumonia was suspected; but later pyelitis developed.

From this time until the latter part of October, the course was fairly smooth; the child then had a temperature for about nine days.

Upon admission, the child weighed 8 pounds 3 ounces. She gained weight rather slowly but steadily, attaining her best weight of 23 pounds 2 ounces in January, 1936. From this time until operation the weight remained stationary.

The laboratory work was essentially negative except for the presence of albumin and red blood cells in the urine.

In February, 1937, her condition was such that we performed a transplantation of the right ureter by the matchstick technique. The child's condition was poor following the

operation and she appeared toxic and dehydrated. The pulse was poor and respirations rapid. She expired on February 28, 1937, from shock and ileus.

X-ray examination soon after admission showed normal function of both kidneys.

On March 24, a plastic repair of the epispadias was done. The child's condition follow-

FIG. 1. Pyelograms taken shortly after both ureters were transplanted into rectum. These show definite nephrotic changes as compared to pyelograms taken before operation.

Autopsy corroborated the diagnosis of paralytic ileus. The implant seemed normal and there was no definite peritonitis.

CASE VII. R. M., male, age 13, was admitted to the hospital on March 5, 1937, with chief complaints of incontinence of urine and a deformity of the genitalia (epispadias). The patient had never any control of urination.

Past medical history and family history were both essentially negative.

Physical examination revealed a suprapubic scar from a previous operation for correction of the epispadias. The body of the penis was adherent to the abdominal wall and the urethral opening was very close to the abdomen. There was no body to the penis, scar tissue covering it and only the glans protruding.

ing this operation was good. He ran a slight temperature for a few days; some infection about the penis cleared. The incontinence still remained. As the child's general condition gradually improved, on April 24 both ureters were transplanted by the matchstick technique.

Comment. Among these four patients one had learned to live a solitary life and preferred life as it was to the chance of death, and the other three were children. Personally, I am glad the little girl and boy died. The other child, 13 years of age, has been turned into a fairly normal boy, although the penile condition should be corrected at some future time. There is now definite nephrosis of both kidneys,

which may prove fatal in the event of serious illness. In spite of this, he is free from pain, he can control his rectum; he

lesion seems to heal if the more involved organ is removed. The tuberculous cystitis also heals but often the bladder is so con-

Fig. 2. Pyelograms taken ten months after bilateral transplantation of ureters into rectum. These show marked nephrosis. The end result will be death due to infection or renal insufficiency. At present there are no physical signs of either and the boy is apparently perfectly well and symptom-free. Prognosis is bad.

voids and defecates four times daily and has perfect night control. He is a favorite now with the other children. The mortality rate was 66⅔ per cent and the morbidity cannot at present be ascertained. Certainly the surviving patient's kidneys have suffered considerable damage as a result of the transplantation and only time will tell what the outcome will be.

TUBERCULOSIS OF THE BLADDER

There are a series of patients who have tuberculosis of the kidneys and bladder upon whom nephrectomy is indicated. As a rule, if the opposite kidney is involved the

tracted that the subjective symptoms of frequency, urgency and pain are as bad or worse than before nephrectomy. This is due to the healing fibrosis of the bladder wall limiting bladder capacity. In these cases I unhesitatingly transplant the ureter of the remaining kidney into the bowel. My results have been very gratifying. Out of four cases, three are alive and well. Two were operated upon ten years ago, and the third one year ago. In my opinion, transplantation is definitely indicated in this type of case.

CASE VIII. A. W. A., male, age 45, was admitted on January 20, 1928. The diagnosis

was left renal tuberculosis and tuberculous cystitis, confirmed by complete cystoscopic study. All the laboratory reports showed tubercle bacilli in the urine. On January 25, a left nephrectomy was done. The patient was discharged February 18, with a healing tuberculous cystitis.

He returned to the hospital on April 28. The bladder was lavaged and over-distended; its capacity was about 1 ounce. Tubercle bacilli were found in the urine. The same treatment was again carried out on April 30 and May 8 when few tubercle bacilli were found in the urine. Some days there would be three plus albumin, other days no albumin. All urine was highly acid and loaded with blood cells. The urea was 12.2; creatinine 1.2; sugar 115; the Wassermann negative. Cystoscopic examination showed the bladder very much improved over the condition at the time of operation. The left ureter was obstructed 3 cm. above the bladder, with a tuberculous fistula at the site of operation. The tuberculous cystitis was improving.

On May 12, the patient was discharged with a diagnosis of cystitis, tuberculosis of the stump of the left ureter and sinus in the old wound.

On November 16, he was again admitted with a diagnosis of tuberculosis of the bladder. The capacity of the bladder was about 1 ounce. No tubercle bacilli were obtainable from the urine. The patient's chief complaint was frequency of urination. The tuberculous fistula in the flank was healed.

On November 19, the right ureter was transplanted into the rectum, according to the more elaborate Coffey method. For a few days following the operation the patient had some elevation of temperature and some rapidity of the pulse. The catheter came out on the tenth day and the rectum acted as a perfect reservoir for the urine. There was no excoriation around the anus. The patient was perfectly comfortable, emptying the rectum about three times a day and once at night. There was no need for cathartics.

On January 1, 1931, the patient was perfectly well, doing hard, manual labor. His rectum accommodated from 8 to 12 ounces of urine which he had no difficulty retaining.

CASE IX.   J. S., male, age 37, was admitted to the hospital on June 6, 1928. The cystoscopic diagnosis was tuberculosis of the bladder,

left renal tuberculosis, and left tuberculous epididymis. Many tubercle bacilli were found in the urine.

On July 2, a left nephrectomy was done Section revealed caseocavernous tuberculosis. Examination on July 14 showed no evidence of tuberculosis in the lungs.

On August 9, a radical operation was done on the right testicle and vas; the left and the right seminal vesicle and both epididymi were removed. Section of all tissue revealed tuberculosis. The albuminuria persisted during the patient's stay in the hospital but cleared up before he was discharged, and the tubercle bacilli disappeared from the urine. Following the nephrectomy and the radical removal of the vesicles and epididymi, the patient was discharged in fair health.

On January 10, 1929, he was again admitted to the hospital with a diagnosis of tuberculous cystitis. He was studied cystoscopically and found to have a very badly contracted bladder, which could not be distended with over 1½ ounces of water without great pain.

The right ureter was transplanted into the rectum by the Coffey catheter technique and the appendix removed on January 14. There were no tubercle bacilli in any of the specimens of urine. Following the operation he had an elevation of temperature for several days. The catheter came away on the fourteenth day and the rectum acted as a reservoir for the urine. The patient made an uneventful recovery and was discharged on February 7, 1929.

On June 6 he reappeared and a diagnosis of possible pulmonary and renal tuberculosis was made. However, all laboratory findings and the x-ray of the chest were negative.

On October 22, x-ray findings still showed nothing suggestive of tuberculosis. At that time the vertebrae were x-rayed for possible tuberculosis of the bone, but this also was negative. The red blood cells were 4,720,000; white blood cells 5,000; polymorphonuclears 68 per cent; lymphocytes 26 per cent. The urea nitrogen was 21.4; sugar 90; the Wassermann negative.

The patient was voiding three or four times a day through the rectum. No cathartics were needed for bowel movements and he was able to hold from 8 to 10 ounces of urine in his rectum comfortably. There was no excoriation around the anus.

In December, 1930, the patient returned to the hospital for a uroselectan observation. The plates were very poor but sufficiently distinct

eighteenth. Autopsy revealed a healed normal transplantation, but the patient died of a general miliary tuberculosis.

FIG. 3. Pyelogram showing bilateral tuberculous pyonephrosis prior to treatment by tuberculin. One year after this picture was taken the right kidney was removed.

FIG. 4. Right kidney has been removed. Left kidney is markedly nephrotic. The bladder can be seen as a small calcified body in the pelvis. Capacity a few c.c. Constant dribbling. Still tubercle bacilli in urine. Pictures taken just before transplantation of left ureter into rectum.

to show a perfectly normal pelvis with some slight dilatation of the ureter. He was in perfect health and doing manual labor.

CASE X. M. M., male, age 65, came to the hospital on March 1, 1935, with pulmonary tuberculosis and a discharging sinus from both testes, accompanied by frequency, urgency and painful micturition.

Examination revealed a right tuberculous caseocavernous nephrosis, marked tuberculous involvement of the bladder, pulmonary involvement, and a chronic myocarditis. The left kidney and ureter were normal. After careful study I decided to reverse the treatment. On March 8, I carefully transplanted the good ureter from the left kidney into the bowel. The patient made an uneventful recovery from the transplantation. As the right kidney was more or less completely destroyed, virtually all of the urine came through the rectum with much relief from the bladder symptoms. It had been planned to remove the tuberculous right kidney, ureter, and epididymi at a later date. On the fifteenth, seven days after the transplantation, the patient began to run an elevated temperature and pulse and died on the

CASE XI. D. W., female, age 8, was admitted to the hospital on January 4, 1937. The chief complaints were pain over the lower abdomen and hematuria of two weeks' duration. Eighteen months previous to this admission the child had been brought to the hospital and a diagnosis of bilateral tuberculosis and tuberculous cystitis was made. The child was inoperable at that time and had been returned home under the care of the family physician who was instructed to place her upon a rigid antituberculous régime and to use Koch's old tuberculin as treatment. After fourteen months, she was readmitted to the hospital. A bilateral pyelogram was made, the right kidney was found to be badly involved and almost functionless and was therefore removed. An uneventful recovery ensued and at the time of her discharge the tubercle bacilli had disappeared from the urine. The caseocavernous type of tuberculosis was noted.

Readmission was necessary because of pain, frequency and constant dribbling. Physical

examination was essentially negative, but x-ray examination by oral urography indicated an advanced pyelonephrosis and a small con-

A year later she returned to the hospital for a checkup. Her weight was 62 pounds, a gain of over 12 pounds since the original operation.
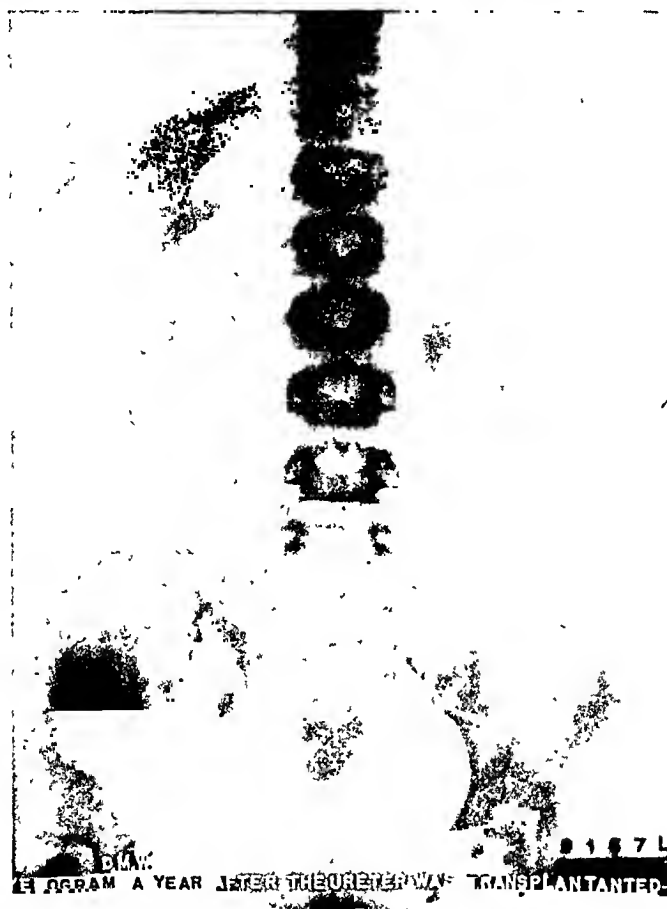
FIG. 5. Fairly normal pyelogram taken a year after transplantation of the left ureter into rectum. Note the difference between this and pyelogram in Fig. 4. This seems to be a fairly normal kidney. No tubercle bacilli in urine. No symptoms.

tracted bladder with a fine calcification of the bladder wall. A few tubercle bacilli were found in the urine. It was decided that this bladder could not be improved by any form of treatment and that the left ureter should be transplanted into the rectum in spite of the fact that there was indication of some active tuberculosis in the left kidney, with the possibility that the ureter might be involved. On January 27, the left ureter was transplanted and the abdomen was closed in layers. Strangely enough, the ureter looked perfectly normal. Following the operation the patient ran a temperature from 101 to 103°F. for nine days; eighteen days later, on February 24, she was out of bed and was sent home greatly improved.

There was no elevation of temperature; her appetite had very much improved; about seven watery stools were passed each day, but there was no incontinence except with coughing or sneezing. She had one bowel movement at night which was watery and which contained little fecal material. Slight excoriation had developed around the anus. Food did not stimulate defecation reflex except in the morning. The pyelogram showed marked improvement in the left kidney.

Comment. The two cases seen in 1930 were adult males, both laborers. They both still work at hard labor; their rectums act satisfactorily as bladders and they are with-

out symptoms. They have perfect control and never wet the bed; they have about four bowel movements daily and are never required to take a laxative.

The third case needs a little comment. I thought it would be good treatment to get the good ureter transplanted before attempting removal of the balance of the active tuberculous urogenital tract. Had I succeeded the idea would have been unusual and the result brilliant. The development of the miliary tuberculosis following the operation cannot be attributed to squeezing tubercle bacilli into the blood stream because no tuberculous tissue was handled. This seems to favor Henline's reasoning that a miliary tuberculosis following removal of a tuberculous kidney is not due to the dissemination of the bacilli by handling. The operation was perfectly successful.

The last case, the little girl, had renal tuberculosis with cystitis, and when she was first seen any surgical procedure was out of the question. An antituberculous régime and Koch's old tuberculin improved her condition in a year, and the worse kidney was then removed. After the operation, the bladder symptoms were as bad as ever; we did not know whether this was due to the infection from the remaining kidney or from the contraction healing of the bladder. Finally the child became totally incontinent. Transplantation of the ureter was decided on, even though this organ might be the seat of a tuberculous process. The operation was a perfect success and today the child has gained 35 pounds, is perfectly continent while awake but does wet the bed once nightly. She is a happy, contented girl and is advancing rapidly at school. I believe as she grows older she will learn how to use her rectum as a bladder at night.

Certainly, without transplantation of the ureters this child would have gradually developed a stricture of the ureter, dilatation of the ureter, a continuation of her renal tuberculosis and an early death from urogenital sepsis. The pyelogram taken a year after the tuberculosis gives no reason to believe that this kidney is not healed and that the rectum will not be a perfect reservoir for the urine.
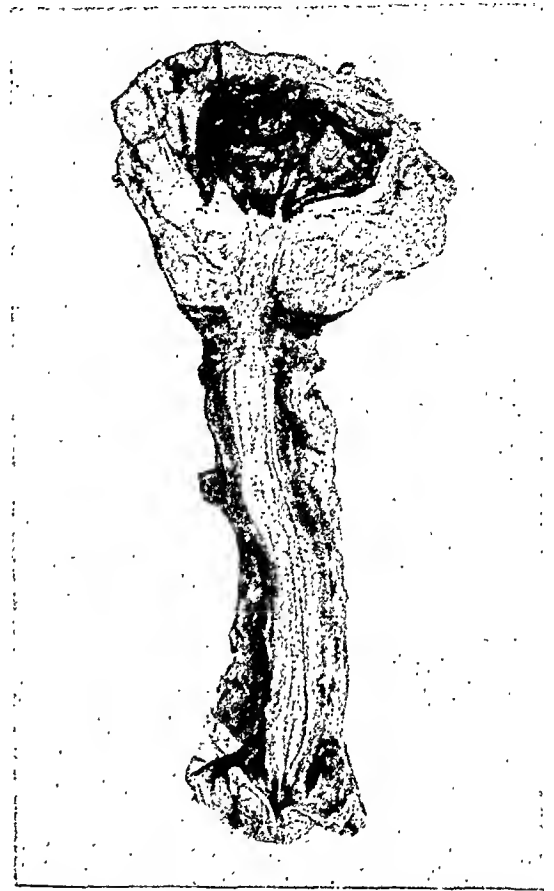
Fig. 6. Bladder and penis in Case xii. Postmortem specimen showing the contracted interstitial cystitis.

INTERSTITIAL CYSTITIS AND CONTRACTED BLADDER

One of the indications for transplantation of the ureter is interstitial cystitis in which the frequent, painful micturition is so burdensome as to make life almost unbearable. The following case had been under the observation of Dr. Braasch at the Mayo Clinic for a number of years. Everything possible in the way of palliation for the interstitial cystitis and contracted bladder, including resection of the presacral nerve, had been done, without affording much relief. Transplantation of the ureters into the sigmoid with later excision of the bladder was therefore advised. Dr. Braasch

referred the patient to me because he wished to be nearer home during his hospitalization.

CASE XII. C. A., male, age 34, had as his chief complaints burning on urination and dysuria.

The patient was in his usual good health until the age of 19, at which time he contracted gonorrhea. He had a discharge for a period of one month. About two months later, he fell about 45 feet from a building and landed partly on the perineum. After a few months he began to have difficulty in urination, frequency (several times a day) and dysuria. This continued gradually to get worse. Three years before I saw him, at the age of 31, he had had a presacral ganglionectomy at the Mayo Clinic. This operation, he stated, aided him for about six months, but the dysuria and frequency reeurred. In February, 1938 he underwent another operation at the University of Pennsylvania Hospital (the nature of which was not determined). This gave him no relief. He had no diseharge or hematuria.

The pain on urination was a dull, heavy, bearing-down one which radiated to the genitalia.

The past medical history was essentially negative except for a history of enuresis at the age of 12. Besides the above operations, a tonsillectomy had been done.

Physieal examination revealed tenderness present in the midline over the lower third of the abdomen. An inguinal lymphadenopathy was present, and the prostate gland was enlarged.

The blood picture was essentially negative, as was the Wassermann. The blood sugar was 60 mg. per 100 c.c. and the blood urea nitrogen 18.9 mg. A catheterized specimen of urine showed albumin, a moderate amount of sugar and a few red and white blood cells.

On x-ray examination, there was no evidence of opaque calculi, but an amorphous calcified density was present low in the right pelvis which might have been in the bladder or adjacent to the bladder. Intravenous pyelogram showed satisfactory kidney function and normal morphology.

The temperature, pulse and respirations were normal. The blood pressure was 120/68. In the hospital, he still suffered as he had upon admission and he also appeared quite nervous.

On July 23, ten days after admission, the diagnosis of interstitial cystitis having been verified, he was operated upon. The bowel was packed away and peritoneal adhesions were found anchoring the sigmoid colon to the left pelvic wall. These adhesions were loosened. The left ureter was found, cut close to the bladder and then transplanted into the bowel. A cigarette drain was inserted and the abdomen closed in layers.

The patient's condition was fairly good immediately following operation. The temperature, pulse and respirations remained normal and there was a total drainage of 22 ounces the first day. However, the next day, the patient's temperature rose to 101 degrees, his pulse rose and he complained of abdominal pain and "gas pains." On the third day, hiccoughs developed and he vomited on several occasions. These symptoms continued, his pulse became irregular, and on the fifth day he became irrational. He died on July 31, eight days after operation.

Autopsy. It was evident that the death was not due to a meehanical obstruetion but to paralytic ileus. The upper two-thirds of the jejunum were mueh dilated, but there was no evidenee of peritonitis exeept the mechanieal evidenee that would go with a paralytie ileus. Nothing could be eultured from the peritoneal fluid.

Comment. This case brings out several very important facts. The first, of course, is the definite difficulty that may be encountered in ureteral transplantation secondary to former abdominal procedures. These procedures, particularly pelvic ones, may easily spoil an operation of this type and must be heavily weighed in making a decision. Second, the regeneration of the presacral ganglion with return of pain six months after ganglionectomy is a valuable observation. How often does this happen? If this can happen once, may it not happen more often than we think? Can we then consider that this surgical procedure must be a more carefully selected one? Did this operative procedure plus the regeneration have anything to do with the irritability of the bowel during operation and the paralytic ileus afterward? Is it not

possible that preliminary transplantation of the ureters instead of ganglionectomy in these cases of interstitial cystitis may not be the proper procedure?

MORTALITY AND MORBIDITY

All told, I have operated on thirteen patients, with sixteen separate operative

Fig. 8. Regenerated presacral nerve. Post-mortem specimen.
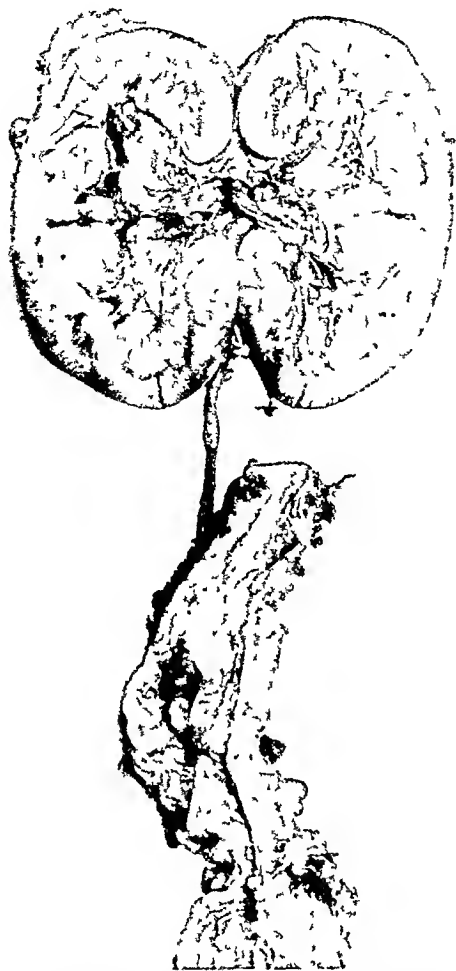
Fig. 7. Right kidney and ureter showing transplantation of ureter into bowel. Post-mortem specimen.

Probably the only possible chance for relief that this boy had from the beginning was transplantation of the ureters. Even though the presacral ganglionectomy had relieved him of his pain, it could never relieve him of his interstitial cystitis nor his contracted bladder; it is doubtful if such a condition is ever curable and if it is curable, the final condition of the bladder will be such that the frequency of urination will be as uncomfortable as is that frequency and urgency which we obtain in the healed contracted tuberculous cystitis. This, then, is a definite indication for transplantation of the ureters.

procedures and seven resultant deaths. Two of these cases were exstrophy of the bladder operated on successfully for other men at other hospitals. The ureters were transplanted separately, making a total of four operative procedures. The patients left the hospital alive, with their rectums functioning. The end results in these two cases is unknown to me.

This report deals only with the cases of which we have permanent records and have been able to follow. There are eleven cases in this series with twelve operative procedures and seven deaths. In several of these cases the operative procedure has been a success but the patients have died of conditions other than those caused directly by the transplant operation. Yet I consider them as transplant mortalities.

All deaths have been autopsied (in itself rather an unusual accomplishment). From a study of these reports, it is my belief that infection does not play such an important rôle in our failures if we are

careful in our suturing and have no bowel or urinary leakage into the peritoneal cavity after operation. Two of the cases had some localized peritonitis at autopsy, which the pathologist felt had nothing to do with the cause of death, and which I believe would have been completely controlled had the patients survived other conditions. As it is a virtual impossibility not to contaminate in some degree the operative field in any of these operations, it can be seen by the study of these few cases that peritonitis per se is usually not due to soiling at the time of operation. Since none of our anastomotic connections had leaked, peritonitis was not the principal cause of death in any of our fatalities.

All of us who have done experimental transplantations upon dogs have found that most of the dogs died of peritonitis. This I think is due to the fact that it is very difficult to do perfect transplantations on these animals because of anatomic difficulties. I agree with Hinman that the elaborate aseptic technique of Higgins is not necessary for the prevention of peritonitis in the human. I do not believe that it makes any particular difference whether or not there is slight soiling at the time of the operative procedure because it is almost a physical impossibility under any form of technique, even that of Higgins, to do this operation without some contamination of the field.

The most frequent cause of death in the series was ileus. Three patients died of this condition and one other died of definite obstruction. In Case I, a single large band of adhesions, unrecognized either before or at operation, caused total obstruction of the bowel postoperatively. This was probably due to bowel handling at the time of operation. Had I been as careful as I should have been and elicited the real story of this man's constipation prior to operation, I would probably have been suspicious of some such condition and might have had a gastrointestinal study made prior to the transplantation. A simple severance of this single band of adhesions

would have prevented an otherwise successful transplantation of the ureters from becoming a fatality. Since this experience, all patients upon whom transplantations are planned are given the benefit of a fluoroscopic gastrointestinal survey.

If we stop to consider just what we do in a transplantation I believe it will be easily understood why ileus has been responsible for so much trouble. Intestinal peristalsis is necessary to prevent ileus and many things are done during this procedure to interfere with it. I also believe that our non-residue diets and our frequent enemas in the preparation of these patients for operation is bad, as there is no doubt that atony of the bowel frequently results because of an empty intestine. The stomach should be empty prior to surgery and so should the sigmoid, but it is a desirable thing ordinarily speaking, to have an intestine partially filled. The packing off of the bowel, the occasional difficulty with straining and evisceration requiring too frequent manipulation of the gut, the clamping of the sigmoid, and the length of time that it requires to complete these surgical procedures, are all predisposing factors in ileus.

What can we do then to prevent ileus? First, the simplest technique possible; second, packing the bowel from the field of operation should be done gently, taking such precautions as to prevent retching and straining, so that repacking does not become necessary. One of these patients had to be repacked four times (Case XII) because of this condition, and I knew that he probably would have a paralytic ileus. All adhesions that bind down the intestine should be carefully freed; all raw surfaces should be covered; if drains are left in, the omentum should be replaced between the small bowel and the drain insofar as possible, and the drain should be placed so that it is not surrounded by coils of intestine.

In two of these patients who succumbed to ileus there had been previous lower abdominal surgery. In both cases the

sigmoid was bound down by dense adhesions. The freeing of these adhesions added to the time of operation and made it much more difficult fully to expose and transplant the ureters. In neither of these cases was there any leakage and only a little local peritoneal irritation. Previous abdominal surgery must therefore be carefully considered when this type of surgery is planned.

One patient died a heart death, probably due to the length of his anesthetic and his operative procedure, but this patient might possibly, had he lived, succumbed to a peritonitis, this being the only case which seemed to have any degree of peritoneal involvement. Prior to the autopsy there was no reason to believe that he had any definite peritoneal involvement.

Case vi died as much from operative shock as from the ileus that was present.

Case v died as a result of an acute obstructive suppurative urethritis and pyelonephritis. A stitch tied too tightly in the bowel completely constricted the left ureter. Several weeks before the right ureter had been successfully implanted. This is definitely an error of operative technique which should not have occurred.

Case x died of a miliary tuberculosis which started seven days after his ureter had been successfully and correctly transplanted. Although the death is recorded as a transplantation death, the transplant site was found to be perfectly normal. There is no doubt that the miliary tuberculosis was the cause of death. Had I this case to do over again I would probably have removed the right tuberculous kidney, the vesicles and epididymi prior to the transplantation and my patient would probably have died of a miliary tuberculosis before I would have had an opportunity to have attempted transplantation. This case is additional evidence supporting Henline's reasoning that miliary tuberculosis is not due to the squeezing of tubercle bacilli into the circulation when a nephrectomy is done for this disease. Here no tuberculous tissue was handled at any time and for that reason tubercle bacilli could not have been squeezed into the blood stream.

A careful study of these seven deaths with their autopsies reveals some very interesting facts. There is no doubt in my mind that quite a number of these cases might have been successful transplants had the operator had more transplantation experience. Certainly peritonitis in the human being should very seldom be the cause of failure. Ileus might be prevented by more careful preoperative preparation, by more gentle handling of the abdominal contents at the time of operation, and by a very simple technique such as the Hinman or the Match-Stick technique, as I call it. All of these operations follow the general principles as advocated by the experimental and clinical experiences of Coffey and Mayo. Leakage certainly has been no factor; shock and cardiac failure and miliary tuberculosis as complications are rather difficult to prevent.

A short review of the successful cases reveals some very interesting facts. Concerning peritonitis as a result of contamination at the time of operation: the most successful case, Case vii, due to lack of coöperation, at operation had a rectum with considerable feces in it. This was washed out as thoroughly as possible before the bowel was opened but in spite of everything that could be done there was contamination to such a degree that I placed drains down to the site of the transplants. He made an uneventful recovery. No doubt there was some localized peritonitis for a few days, but nature took care of it.

The more elaborate the technique, the greater is the chance for trouble. Most of my surviving patients had operations which took less than an hour. In all but two of these cases that died the operative procedure lasted over an hour. From my very small experience I would feel that, all other things being equal, the ureters should be transplanted separately. It ought not to take a skilful man longer than a half hour to transplant one ureter if there are no

adhesions and if he uses a simple technique, such as the Mayo modification of the Coffey, the matchstick, or Hinman technique. It is my belief that the matchstick

the ureter is a little better than the Hinman technique with the ligature around the ureter cut after it is in the bowel. At least
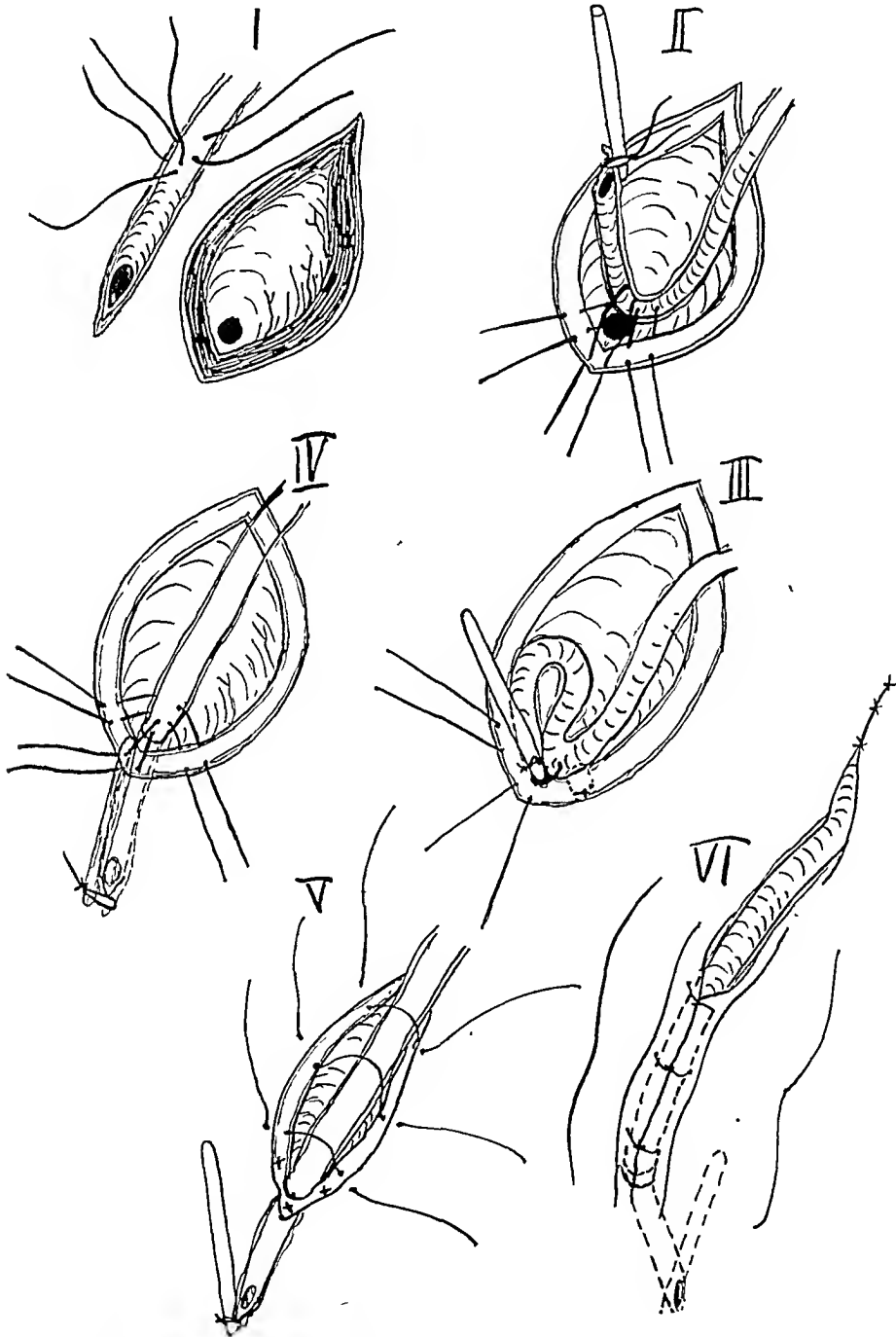
FIG. 9. 1, sutures are placed in ureter and sigmoid is opened. 2, matchstick tied to end of ureter and sutures placed to hold ureter in bowel. 3, inserting free tip of ureter into bowel. 4, tip of ureter in lumen of bowel sutures now tied. 5, sutures placed in bowel to hold ureter against mucosa and underneath muscularis. 6, wound in bowel closed.

nique. It is my belief that the matchstick technique with the wide open oblique cut of

I believe that the end of the ureter should be left wide open with an oblique opening

instead of tying it off as Hinman does. However, the Hinman modification of Coffey's technique is perhaps the best yet. I believe that if his little adaptor were fastened to the cut end of the ureter in the same way as I fasten the matchstick his results would be even better.

TECHNIQUE OF OPERATION

The patient is prepared for several days with simple cleansing enemas. Forty-eight hours before operation an ounce of castor oil is given. About six hours before operation, the bowel is carefully irrigated with plain water. As soon as the abdomen is opened and the bowel is packed away, the anesthetic of choice being spinal, the sigmoid is clamped and the bowel is again carefully washed out with water until the washing becomes clear. If there are any large pieces of feces in the bowel at that time they are flushed out through a proctoscope. The ureter to be implanted is then dissected free without any attempt being made to strip it of its periureteral fascia and fat. It is cut off as near the bladder as possible. The bowel is then prepared according to the Coffey technique. Sutures are placed very much in the fashion that Hinman places his, first three sutures into the ureter through the submucosa and muscularis. (Fig. 9.) The ureter is then obliquely cut after it has been fitted to the incision in the bowel, leaving about an inch to protrude into the lumen of the bowel. The bowel is blown up very gently with air and the mucosa is punctured with the cautery. Through this opening the ureter and the matchstick are plunged into the lumen of the bowel and manipulated until the matchstick disappears within the lumen when the sutures are tied holding the ureter to the submucosa and the muscularis. It may be advisable to put in one or two other sutures to hold the ureter in its bed as per the Hinman technique. The muscle is closed, usually with interrupted sutures over the ureter; one must make sure not to constrict the ureter when tying the sutures. A drain may or may not

be placed in the cul-de-sac, depending upon the operator's judgment. This is a simple speedy technique and in my opinion offers the best chance for success.

All operations in this series were done by the Coffey catheter, the Coffey-Mayo, or the matchstick technique. Of those that survived, two were done by the Coffey catheter, one by the Coffey-Mayo and three by the matchstick technique.

CONCLUSIONS

Transplantation of the ureters into the bowel as an operative procedure should be considered only when a condition exists which makes life sufficiently miserable that death is usually to be preferred. The operation, even with the simplest efficient technique, in the hands of the most skilful, is not without a very high mortality and morbidity. It is rarely indicated in cancer of the bladder and vesicovaginal fistulae. It is more frequently indicated in the contracted bladder due to the healing of a tuberculous cystitis, and in this class of patient there is a low mortality with very satisfactory symptomatic results. It is also indicated in intractable and incurable cases of interstitial cystitis. It is a valuable procedure in exstrophy of the bladder and epispadias, but should never be used in the adult exstrophy without a complete explanation of both its mortality and morbidity and should be an operation elected by the patient.

Note. Since the completion of this paper a new technique has been described by Farrell and Lyman (*Surg., Gynec. & Obst.,* 66: 657, 1938). This technique is described as an aseptic uretero-intestinal anastomosis. The operation was done on seven dogs and all survived. At the end of six months all but two dogs were killed and autopsied. The technique is modeled after the Eck fistula, an anastomosis of the portal vein and the inferior vena cava. While the procedure has given good results in this series of dogs, it apparently has never been applied clinically. The technique seems to be rather difficult and time consuming and

I do not like the blind use of the silk ligature to create the opening in the ureter and bowel. I can see no advantage over the Higgins technique, and believe this new one to be even more difficult and complicated.

REFERENCES

BEER, E. Total cystectomy and partial prostatectomy for infiltrating carcinoma of the neck of the bladder. *Ann. Surg.*, 90: 864 (Nov.) 1929.

COFFEY, R. C. Completed aseptic technique for the implantation of the ureter into the large bowel. *Surg., Gynec. & Obst.*, 45: 816 (Dec.) 1927.

COFFEY, R. C. Transplantation of the ureters into the large intestine. *Surg., Gynec. & Obst.*, 47: 593, 1928.

COFFEY, R. C. Bilateral submucous transplantation of the ureters into the large intestine by tube technique. *J. A. M. A.*, 93: 1529 (Nov. 16) 1929.

COFFEY, R. C. Cystectomy with bilateral transplantation of the ureters in one operation. *Northwest Med.*, 29: 128, 1930.

COFFEY, R. C. Transplantation of the ureters into the large intestine. Submucous implantation method. Personal studies and experiences. *Brit. J. Urol.*, 3: 353–428, 1931.

HESS, E. Pyelocystostomy (pyelocystostomosis) in crossed renal dystopia. *J. Urol.*, 22: 667–683, 1929.

HIGGINS, C. C. Aseptic uretero-intestinal anastomosis. *Surg., Gynec. & Obst.*, 57: 359–361, 1933; *Am. J. Surg.*, 22: 207–209, 1933.

HIGGINS, C. C. Aseptic uretero-intestinal anastomosis. *J. Urol.*, 31: 791–802, 1934.

HIGGINS, C. C. Transplantation of the ureters into the rectosigmoid. An experimental and clinical consideration. *J. Urol.*, 37: 90 (Jan.) 1937.

HIGGINS, C. C. Cystectomy and transplantation of the ureters into the bowel for carcinoma of the bladder. *Surg., Gynec. & Obst.*, 66: 549–555 (Feb. 15) 1938.

HINMAN, F. Simple seven suture method of bilateral uretero-intestinal implantation; report of twelve cases. *Surg., Gynec. & Obst.*, 61: 802–815, 1935.

HINMAN, F. An experimental study of uretero-intestinal implantation. *Surg., Gynec. & Obst.*, 62: 909–917, 1936.

HINMAN, F. A critical study of the different principles of surgery which have been used in uretero-intestinal implantation. *Surg., Gynec. & Obst.*, 64: 313–363, 1937.

KIRWIN, T. J. A study of ureteral implantation with a description of a new procedure. A preliminary report. *Am. J. Surg.*, 8: 1, 1930.

KIRWIN, T. J. Clinical value of experimental ureteral implantation. *Am. J. Surg.*, 23: 14–29; 42 (Jan.) 1934.

MATHÉ, C. P. Diversion of the urinary stream by ureteral transplantation. Its indications and ultimate results. *California & West. Med.*, 34: 97 (Feb.) 1931.

MAYO, C. H., and WALTERS, W. Transplantation of the ureters into the rectum; end-results in 35 cases of exstrophy of the bladder. *J. A. M. A.*, 82: 624–626, 1924.

MAYO, C. H., and HENDRICKS, W. A. Exstrophy of the bladder. *Surg., Gynec. & Obst.*, 43: 129–134, 1926.

TREATMENT OF INTRACTABLE SCIATIC PAIN DUE TO PROTRUDED INTERVERTEBRAL DISCS*

WINCHELL McK. CRAIG, M.D.

Section on Neurologic Surgery, The Mayo Clinic

ROCHESTER, MINNESOTA

SCIATICA, or pain over the posterior aspect of the hip, thigh, and leg, is a very common complaint, producing both acute and chronic discomfort. This pain is not only characterized by its resistance to treatment, but also by the fact that it can be a symptom of so many disease entities. The sciatic nerve is, of course, the longest nerve in the body; it arises from the primary divisions of the fourth and fifth lumbar nerves and the first and second sacral nerves, and it is subjected to trauma, strain, distortion, infection, and compression. For this reason, many underlying causes produce irritation to the nerve and its component parts, intraspinally, within the pelvis, and externally. The term "sciatica" evidently was evolved from the term "ischias," which was used by the ancients in referring to all painful conditions of the thigh and hip; as a consequence, there is a tendency to include many irrelevant conditions under this title. For that reason the term "sciatica" should be used to signify a symptom without any indication of the underlying cause.

Formerly it was customary to divide all cases of sciatic pain into a primary and secondary group. In the primary group were placed all the so-called idiopathic cases, or those in which the pain was apparently due to pathologic processes involving the nerve elements primarily. In the secondary group were placed all cases in which the underlying cause could be traced to a lesion of the contiguous bony or muscular structures and in which the nerves were involved secondarily. Of recent years, the primary group has dwindled as our knowledge of the many and diverse lesions of the bony and muscular structures which produce irritation of the sciatic nerve has increased.

Ghormley,[9] in discussing low back and sciatic pain from an orthopedic standpoint, divided the types of conditions and lesions which may be the underlying cause into seven divisions: In the first division he placed acute and chronic strain due to lumbosacral or sacro-iliac lesions. In the second division he placed all types of trauma. Trauma involving the vertebrae, such as fractures of the body, pedicles, laminae, or facets, he considered fairly obvious with regard to the diagnosis because of the history and roentgenologic findings. Trauma involving the joints and producing traumatic spondylosis he also considered fairly obvious. Traumatic conditions involving the intervertebral discs, however, including narrowing and avulsion of the disc and rupture of the nucleus pulposus, are sometimes difficult to diagnose and almost always require the combined efforts of the orthopedist, neurologist, and neurosurgeon before the proper diagnosis and treatment can be carried out. Under infections Ghormley included infectious arthritis, fibrositis, typhoid fever and tuberculous spine. Metabolic and senescent conditions included hypertrophic changes and osteoporosis with pathologic fracture. Congenital anomalies included spina bifida, sacralization of the fifth lumbar vertebra or lumbarization of the first sacral vertebra, and anomalous facets; these are conditions that can be demonstrated in the roentgenograms. Neoplastic conditions he divided into the benign and malignant.

Under benign neoplasms were osteoma and osteochondroma, giant-cell tumor, and hemangioma; these are usually evident in the roentgenograms. Under malignant tumors was metastasis from other malignant lesions in the body; this is sometimes difficult to diagnose. Myeloma and primary osteogenic sarcoma are evident in the roentgenograms. Ghormley's last classification included such neurologic conditions as tumor, inflammation, and degeneration of the spinal cord.

In addition to the orthopedic conditions which may produce sciatic pain are the constitutional or systemic causes, which include such conditions as diabetes, syphilis, and gout, and forms of toxic neuritis which develop following the absorption of alcohol, lead, or arsenic. Shelden has called attention to the presence of either primary or metastatic pelvic tumors which may produce severe sciatic pain and which can only be identified by the routine use of rectal examinations.

It is apparent, then, that the underlying cause of sciatic pain is quite frequently discovered during a comprehensive physical examination. The general examination, including careful observation of motion, tenderness, and the abnormal appearance of the back and lower extremities, together with comparative roentgenograms of the lower part of the spine and sacrum and neurologic examination to evaluate any motor or sensory changes in the muscles subserved by the sciatic nerve, may lead to a definite diagnosis. In a relatively moderate sized group of cases of sciatic pain the cerebrospinal fluid must be examined in order to make the diagnosis. In these cases spinal puncture is carried out with the patient in the prone position, and usually at the fourth interspace; the manometric pressure is taken before, during, and after compression of the jugular veins (Queckenstedt test) to determine the presence or absence of intraspinal pressure producing partial or complete subarachnoid block. The cell count of the cerebrospinal fluid is important in differentiating inflam-matory from other irritative lesions. Estimation of the total protein is likewise important because, in the case of many irritative lesions, the increase in total protein is the only indication of the condition.

Before determining definitely whether the underlying lesion causing the sciatic pain is of such a nature as to require operation, it is sometimes advisable to carry out conservative therapeutic measures, as previously described by Ghormley and myself. These consist of rest in bed with extension, and the use of a sacro-iliac belt, diathermy, and epidural injections. We have observed that patients who, under such conservative management, have an increase in pain either on extension or following epidural injections, usually have an intraspinal lesion requiring laminectomy for relief of their pain.

In recent years attention has been called to the rôle of protruded or herniated intervertebral discs in the etiology of intractable sciatic pain. In many cases in which patients have heretofore been classified as having idiopathic sciatic pain and have been treated by various methods, with or without temporary or palliative relief, sciatic pain has probably been due to this definite intraspinal lesion. Goldthwait, and Middleton and Teacher, in 1911, called attention to the possibility of protrusion of an intervertebral disc into the spinal canal causing compression of the cauda equina and clinical symptoms. Adson, in 1922, and Stookey, in 1928, reported cases of compression of the cervical portion of the spinal cord resulting from protrusion of intervertebral discs. In 1929 Dandy reported two cases in which protrusion of lumbar intervertebral discs had caused paraplegia, with relief following operation; he stressed the importance of trauma in the production of these lesions. Mixter and Barr, in 1934, and Hawk, in 1936, further emphasized protruded discs as being one of the obscure causes of sciatic pain.

In the Section on Neurology at The Mayo Clinic these intradural fibrocartilaginous masses have been observed for many years. As early as 1924, because of a pathologic diagnosis of fibrochondromatosis or intervertebral disc, these masses were included with extradural tumors of the spinal cord. That trauma plays a rôle in the production of protruded intervertebral disc is an accepted fact, as evidenced in the majority of cases by the history. The explanation for this is that the restraining annulus fibrosus is ruptured or weakened by undue stress and strain, following which the nucleus pulposus protrudes or herniates into the spinal canal, with subsequent encroachment on the nerves.

The intervertebral discs, which act as shock absorbers between the vertebrae, are composed of a nucleus pulposus which is encircled by the fibrocartilaginous structure, the annulus fibrosus. When the spinal column is subjected to stress and strain, the disc may bulge in all directions although, normally, it returns to its natural position where it is maintained by the vertebral ligaments, principally the anterior and posterior longitudinal ligaments. Following trauma to the vertebral column, or during unusual stress or strain, the disc, itself, or its center, the nucleus pulposus, may be extruded beyond normal limits and may fail to return to the original position. The protrusions which cause clinical symptoms are those which occur posteriorly, and they evidently occur most often in this location because of mechanical forces at the time of injury to the spine. Most of the injuries occur when the spine is in flexion, which produces undue stress on the posterior portion of the annulus fibrosus, the anterior portion of the vertebral interspace being narrowed. In this manner the compressible nucleus pulposus is compressed backward against an overstretched and weakened annulus fibrosus, and while the latter may not give way the first time, extrusion may result during subsequent injury.

In making a diagnosis of protruded intervertebral disc many factors are involved; heretofore, only those patients who did not respond to other types of treatment and in whose case a definite diagnosis could not be made were suspected of having protruded intervertebral discs. As is true of so many other clinical syndromes, the number of cases in which the diagnosis is made and treatment carried out is becoming greater because of the fact that the condition is being looked for during the examination. As a result, much time is saved and much needless suffering prevented.

The symptoms of protruded intervertebral disc vary greatly. The chief symptom, however, is root pain or a pain which begins within the spinal canal and is projected peripherally to that part of the body or extremity innervated by the nerve. The pain is often described as sharp or shooting, and it may be increased by coughing, jarring, or sneezing. It may be precipitated or increased by putting tension on the nerve, as by bending the neck or back. This is especially true in cases of sciatic pain in which positive Kernig or Lasègue signs are elicited when the roots of the sciatic nerve are compressed by a protruded intervertebral disc. In testing for the presence of a protruded intervertebral disc as the cause of intractable sciatic pain, the Lasègue test is important. It consists of flexing the thigh upon the abdomen, with flexion of the leg at the knee. If the leg is passively extended, pain will be felt along the course of the nerve, inhibiting movement before complete extension of the leg is secured. This test may also be applied in the sitting position, the patient in attempting to extend his leg complaining of an increase of pain in the affected leg which prevents full extension of the knee.

There are, however, no pathognomonic symptoms of protruded intervertebrals dic. Roentgenograms of the spine are of some value in making the diagnosis, as emphasized by Love and Camp, who analyzed fifty cases of protruded intervertebral disc

proved at operation. In fourteen of these cases plain roentgenograms were negative and gave no assistance in making a diagnosis. In one case the lesion was partially calcified. In the early reports of protruded intervertebral disc it was thought that narrowing of the intervertebral space was of diagnostic significance, but the fact that such narrowed intervertebral spaces are evident in cases in which no clinical signs of protruded intervertebral disc can be found and that they have been found to be multiple in so many cases, shows that it is of little significance. Hypertrophic changes and compression fractures are also of little diagnostic value.

The most important roentgenologic phase of the diagnosis of protruded intervertebral disc according to Camp,[3] is interpretation of the lipiodol studies. In many clinics such studies are considered essential in making a preoperative diagnosis of protruded intervertebral disc. The introduction of air into the subarachnoid space of the lower lumbar and sacral spine has, according to Fay and Pancoast, also proved of value in localizing and diagnosing protruded intervertebral discs in some neurosurgical clinics.

In order to localize the lesion and definitely establish a preoperative diagnosis it seems necessary to inject radiopaque oil and examine the patient under the fluoroscope as well as make multiple roentgenograms in different positions. The use of lipiodol indiscriminately in all cases of persistent sciatic pain is not to be recommended; this test should be made a part of the general examination and be used only when the clinical examinations indicate it as necessary.

In a review of our cases of tumor of the spinal cord at the Mayo Clinic, I[5] emphasized the danger of the indiscriminate use of lipiodol, but I indicated that, in properly selected cases, the information gained by its use outweighs any of its dangers or disadvantages. Camp has emphasized the necessity of using sufficient radiopaque oil (5 c.c.) to fill the subarachnoid space at any

desired level in order to visualize small non-obstructing lesions which can be identified by a filling defect. This filling defect assumes a characteristic shape, depending on whether the lesion is extradural, intradural, or intramedullary. Since the majority of protruded intervertebral discs do not produce obstruction and occur in a region where the spinal canal is really large, it is hopeless to attempt to visualize or to locate them by means of only 1 or 2 c.c. of radiopaque oil; such a small amount can easily trickle past the lesion without producing any significant change in contour.

In making lipiodol studies in cases of intractable sciatic pain the lumbar injection of the oil is preferred. After the injection, the patient is placed in a sitting position for a few minutes to allow the oil to descend into the sacral region. Later, on the fluoroscopic table in the prone position, the oil is observed as it flows cephalad under the fluoroscope. In this way defects can be observed, and several roentgenograms should be made immediately. Fluoroscopic examination should also include observations in the prone, oblique, and lateral positions in order to verify the level of the lesion as well as aid in establishing its location to one side or the other. In addition to the outline of the defect, produced by the mass of the protruded disc, significant changes in the shadows of the nerve root may be present at the level of the lesion. These consist of edema of one or more nerve roots which may be localized by broadening of the negative shadow of the nerve root. The surgeon sometimes has difficulty at the operating table in associating the pathologic changes found with the roentgenologic shadows, but Hampton and Robinson have offered a solution for this by calling attention to the fact that the fifth lumbar root and most of the first sacral root are extradural at the lumbosacral junction; for this reason a lesion compressing either of these roots may produce a minimal defect in a contiguous subarachnoid space.

Another condition which may contribute to intractable sciatic pain and which may or may not be associated with protruded intervertebral disc is thickening of the ligamentum flavum. This was first emphasized by Towne and Reichert in 1931. They reported two cases in which there was compression of the cauda equina; at operation, hypertrophy of the ligamentum flavum was removed, with complete relief of clinical symptoms. The ligamenta flava stretch across the posterolateral aspect of the spinal canal between the laminae. These ligaments, which are made up of yellow elastic fibers, are attached to the anterior aspect of the superior laminae and to the posterior aspect of the inferior laminae. The two ligaments of each interlaminar space fuse in the midline and extend laterally to form the posterior margin of the intervertebral foramina. Hypertrophy of these ligaments, either due to inflammation or trauma, narrows the intervertebral canal and may constrict the nerves of the cauda equina or compress the nerve roots opposite the segment. It is evident, therefore, that in the presence of a protruded intervertebral disc any hypertrophy of a ligamentum flavum would increase the symptoms proportionately.

Since the original report, Spurling, Mayfield and Rogers, and Abbott, have called attention to this pathologic condition which can produce intractable pain and other associated clinical evidence of compression of the spinal nerve roots. In the roentgenologic examination following injection of radiopaque oil in the subarachnoid space, hypertrophy of the ligamenta flava becomes evident in narrowing the canal, and this in some cases produces shadows which, according to Camp,[4] are pathognomonic. These lesions are mentioned in passing as contributing to the cause of intractable pain because, at operation, such lesions are removed with the discs, relieving pressure on the nerve roots.

In respect to the diagnosis of protruded intervertebral disc as a cause of intractable sciatic pain, it is evident that no definite clinical syndrome is produced. In an effort to discover a clinical syndrome which would be of diagnostic importance, Walsh and Love recently reviewed a series of 100 cases proved by comprehensive general and neurologic examination and at operation to be protruded intervertebral disc. Some very interesting data were brought out: Protruded intervertebral discs may occur in the cervical, thoracic, or lumbar regions, but the majority occurs in the lumbar region and almost 50 per cent occur at the lumbosacral junction. Walsh and Love found that the majority of protruded intervertebral discs occurs in males (there were seventy-seven males and twenty-three females in their series) and that these lesions occurred predominantly in the fourth decade, the average age of the patients being 40 years, the youngest being 16 and the oldest 77. Of these 100 protruded intervertebral discs, eighty-eight were in the lumbar region. In view of the fact that these are the cases we are most interested in with regard to intractable sciatic pain it was interesting to note that 94 per cent of the patients suffering from protruded lumbar discs complained of sciatic pain. It was also interesting to note that in 83 per cent of these cases there was a history of injury. While continuous and persistent sciatic pain is encountered frequently, in 86 per cent of these cases the pain was intermittent. In addition to the sciatic pain, which was present in 94 per cent of the cases, there was an associated low back pain in 55 per cent which had preceded the appearance of the sciatic pain. In 2 per cent there was an associated femoral extension of the pain.

In cases of tumor of the spinal cord the majority of patients complain of night pain; they are awakened and the pain is relieved by moving about or when they sleep for the rest of the night in a chair. In 31 per cent of these cases of protruded intervertebral disc night pain was present. Another characteristic of the pain of tumor of the spinal cord is its accentuation on coughing, sneezing, or straining; in 39 per

cent of the cases of lumbar protruded intervertebral disc this was also true. In addition to intractable pain, there was paraplegia in 40 per cent of the cases and sphincteric incontinence in 8 per cent.

In attempting to augment the history in determining a characteristic syndrome, the neurologic findings in these eighty-eight cases of protruded lumbar intervertebral discs were interesting. There was a positive Lasègue sign or increase in pain on stretching of the sciatic nerve in 82 per cent of the cases. There was sciatic tenderness in 63 per cent. The Achilles reflex was diminished or absent in 56 per cent of the cases and the hamstring reflexes were diminished or absent in 27 per cent. The patellar reflexes were diminished or absent in only 9 per cent. There was muscular paresis in 26 per cent, muscular atrophy in 9 per cent, and sensory loss in 31 per cent; in only 3 per cent was the neurologic examination objectively negative. However, the examination was negative except for positive Lasègue signs of sciatic tenderness in 16 per cent of the cases. The average duration of symptoms was six years, the shortest duration being one month, the longest thirty-two years. The average duration of low back pain before the development of sciatic pain averaged four years. The cerebrospinal fluid was examined in these eighty-eight cases in an effort to establish the basis for the differential diagnosis, and the determination of total protein seemed to be the only outstanding factor. Total protein was estimated in only eighty-four cases; in 81 per cent of these cases it averaged 40 mg. per 100 c.c. or more, the highest value being 240 mg. In 19 per cent of the cases the total protein averaged less than 40 mg., the lowest value being 20 mg. per 100 c.c.

On analyzing the results of the clinical investigative study in these cases of protruded intervertebral disc, a careful examination elicited evidence of nerve involvement, as indicated by the positive Lasègue sign, sciatic tenderness, reflex changes, paresis, atrophy, or sensory loss in 97 per cent of the cases, the examination being objectively negative in only 3 per cent. The increase in the total protein of the spinal fluid in 81 per cent of the cases indicates that this is a valuable clinical finding.

The treatment of intractable sciatic pain due to protruded intervertebral disc compressing the nerves or nerve roots is surgical in all cases. Temporary and palliative relief may be obtained by conservative orthopedic treatment such as extension, support by means of sacro-iliac belts and braces, and fusion, but unfortunately the relief is only temporary and the irritation of the nerve roots recurs. It is conceivable that in some cases facetectomy, as suggested by Ghormley,[10] would decrease irritation to the nerve root and result in relief. The tendency for the protruded disc to increase in size, however, and the possibility of some hypertrophy of the overlying ligamentum flavum, indicates that the treatment of choice consists of laminectomy with removal of the protruded disc.

Once having localized the lesion by means of fluoroscopic examination and by subsequent roentgenographic examinations with radiopaque oil, a small laminectomy, involving only two spines and laminae, is all that is necessary in the majority of cases. The anesthetic of choice is ether, and it is best administered by the open drop method over the end of a Magill intratracheal tube which has been introduced after the patient has been anesthetized by nitrous oxide and oxygen. After the patient has been anesthetized, he is placed prone on the operating table in such a manner that the prominence of the lumbar and sacral spinous processes is increased. Through a vertical incision, subperiosteal resection of the spinous processes and laminae is carried out. The extent of the laminectomy depends a great deal, of course, upon the location of the protruded disc. In cases in which the protrusion is unilateral it may be necessary to remove the articular facets on that side. The protruded disc may be removed extradurally or transdurally, and again it depends a great deal upon the ease

of exposure whether the dura is retracted to expose the protruded disc or is incised and the disc approached intradurally. Protrusions of the disc can be palpated and the exposure carried out as indicated. When the protrusion has been exposed by retracting the dura mater, the thinned out posterior longitudinal ligament is incised either longitudinally or by means of a crucial incision.

Protrusions may be of two kinds, in that the posterior longitudinal ligament may be so thin that the protruded disc pops into the wound, or the disc being fibrous and adherent, it may have to be curetted from its bed. All protruding tissue that is causing or could cause pressure on the nerve roots or spinal cord should be removed. One of the outstanding features encountered at the operating table is the evidence of irritation on the adjacent nerve roots. They are increased in size and edematous and sometimes are injected, showing that the intractable pain has been caused by irritation of the protruded disc. Following removal of the disc the lipiodol is removed and the wound is closed; usually a Penrose drain is left in place from twenty-four to forty-eight hours. Patients are kept in bed following operation for from ten to fourteen days and, if voluntary urination does not occur by the third day, an inlying urethral catheter is placed for continuous drainage.

The postoperative results in all cases of intractable sciatic pain have been most gratifying. The mortality also is gratifying, there having been no deaths in the entire series. In the literature there are reports of operative cases of protruded intervertebral discs in which fusions have been necessary to relieve subsequent postoperative pain. In none of our cases has this been true.

SUMMARY

Intractable sciatic pain which is persistent, intermittent, and does not respond satisfactorily to any type of treatment should be suspected as being due to a protruded intervertebral disc. In a large percentage of cases a careful and complete examination will elicit certain evidences of underlying pathologic change, although it may be necessary to localize and identify the lesion by means of roentgenologic examinations following the injection of radiopaque oil within the subarachnoid space. The presence of increased protein in the cerebrospinal fluid and the positive Lasègue sign, changes in reflexes, and tenderness in the sciatic nerve, are helpful although not essential in making the diagnosis.

The treatment of choice is laminectomy, with removal of the protruded intervertebral disk as well as a possible associated hypertrophied ligamentum flavum. There were no deaths in a series of 100 cases.

Postoperatively, the patient should be cautioned not to return to work too soon, and for a long period of time no heavy labor should be attempted. As a group the patients suffering from intractable sciatic pain caused by protruded intervertebral discs are permanently relieved of their pain and disability.

REFERENCES

1. ABBOTT, W. D. Compression of the cauda equina by the ligamentum flavum. *J. A. M. A.,* 106: 2129–2130 (June 20) 1936.
2. ADSON, A. W., and OTT, W. O. Results of the removal of tumors of the spinal cord. *Arch. Neurol. & Psychiat.,* 8: 520–537 (Nov.) 1922.
3. CAMP, J. D. Roentgenologic findings in cases of protruded intervertebral disks. *Proc. Staff Mcet. Mayo Clin.,* 12: 373–377 (June 16) 1937.
4. CAMP, J. D. Personal communication.
5. CRAIG, W. McK. The use and abuse of iodized oil in the diagnosis of lesions of the spinal cord. *Surg., Gynec., & Obst.,* 49: 17–28 (July) 1929.
6. CRAIG, W. McK., and GHORMLEY, R. K. The significance and treatment of sciatic pain. *J. A. M. A.,* 100: 1143–1149 (April 15) 1933.
7. DANDY, W. E. Loose cartilage from intervertebral disk simulating tumor of the spinal cord. *Arch. Surg.,* 19: 660–672 (Oct.) 1929.
8. FAY, T. S., and PANCOAST, H. K. Personal communication.
9. GHORMLEY, R. K. Low back and sciatic pain from an orthopedic standpoint. *Proc. Staff Meet. Mayo Clin.,* 12: 394–396 (June 23) 1937.
10. GHORMLEY, R. K. Personal communication.
11. GOLDTHWAIT, J. E. The lumbosacral articulation; an explanation of many cases of "lumbago," "sciatica" and paraplegia. *Boston M. & S. J.,* 164: 365, 1911.

12. Hampton, A. O., and Robinson, J. M. The roentgenographic demonstration of rupture of one intervertebral disk into the spinal canal after the injection of lipiodol. With special reference to unilateral lumbar lesions accompanied by low back pain with "sciatica" radiation. *Am. J. Roentgenol.*, 36: 782–803 (Dec.) 1936.

13. Hawk, W. A. Spinal compression caused by ecchondrosis of the intervertebral fibrocartilage: with a review of the recent literature. *Brain*, 59: 204–224 (June) 1936.

14. Love, J. G., and Camp, J. D. Root pain resulting from intraspinal protrusion of intervertebral discs. Diagnosis and surgical treatment. *J. Bone & Joint Surg.*, 19: 776–804 (July) 1937.

15. Middleton, G. S., and Teacher, J. H. Injury of the spinal cord due to rupture of an intervertebral disc during muscular effort. *Glasgow M. J.*, 76: 1–6, 1911.

16. Mixter, W. J., and Barr, J. S. Rupture of the intervertebral disk with involvement of the spinal canal. *New England J. Med.*, 211: 210–215 (Aug. 2) 1934.

17. Shelden, W. D. The differential diagnosis of neurologic lesions resulting in low back and sciatic pain. *Proc. Staff Meet. Mayo Clin.*, 12: 396–397 (June 23) 1937.

18. Spurling, R. G., Mayfield, F. H., and Rogers, J. B. Hypertrophy of ligamenta flava as cause of low back pain. *J. A. M. A.*, 109: 928–933 (Sept. 18) 1937.

19. Stookey, Byron. Compression of the spinal cord due to ventral extradural cervical chondromas; diagnosis and surgical treatment. *Arch. Neurol. & Psychiat.*, 20: 275–291 (Aug.) 1928.

20. Towne, E. B., and Reichert, F. L. Compression of the lumbosacral roots of the spinal cord by thickened ligamenta flava. *Ann. Surg.*, 94: 327–336 (Sept.) 1931.

21. Walsh, N. M., and Love, J. G. Protruded intervertebral disks as a cause of intractable pain. *J. A. M. A.*, 111: 396–400 (July 30) 1938.

The gravity of a head injury depends on the degree of damage to the brain. While it is important to know whether or not the skull has been fractured, it is far more essential to ascertain how severely the brain has been injured.

From—"A Textbook of Surgery" edited by Frederick Christopher (Saunders).

PIGMENTED NEVI

WITH SPECIAL REFERENCE TO THEIR SURGICAL TREATMENT

Jacques W. Maliniac, m.d.

NEW YORK CITY

LARGE nevi and hairy moles are skin blemishes which are particularly conspicuous when located on the face. They then present a difficult dermatologic problem.

Pathologically they represent a circumscribed increase of skin pigment, often associated with hypertrophy of one or all the cutaneous structures, especially connective tissue and hair.

As a rule, once established they remain permanent, with no tendency to reduction in size. There is often a disposition to an increased growth of hair. The blemish is benign unless constantly irritated, when degenerative changes may set in.

Treatment. When small, the benign growth is easily destroyed by desiccation or by means of a keratolytic substance (trichloracetic acid, etc.). This procedure is inadvisable for flat nevi, even of small size, as it necessarily produces a depressed scar. Excision with fine approximation results in a linear scar which is generally less conspicuous than a round depressed one.

In large nevi, use of the electric needle or a keratolytic substance is contraindicated because of the long time such treatment requires and the conspicuous scarring which is inevitably produced. The disfiguring scar usually requires reparative surgery at a later date. Besides, repeated prolonged treatment is extremely difficult, if not possible, in small children, who represent the majority of these cases.

Radiation therapy has been advocated in the treatment of pigmented nevi. The ease with which it can be applied is responsible for abuses in its use. Authoritative opinion on this matter is clearly expressed by McKee,[1] who states that "X-rays and radium are not indicated in the treatment of naevus pigmentosus. The author tried beta and gamma rays without result."

FIG. I. A, depressed, irregular scar with atrophy of cheek in girl of fifteen, following application of radium for nevus in early infancy. B, condition after repeated partial excision of scar with free full-thickness graft from upper lid to upper end of defect. Subcutaneous insertion of a dermo-fat graft (from abdomen) to raise depressed cheek.

Plastic Repair. Proper mental hygiene requires that all those affected by this extensive skin blemish should be treated at an early age. Replacement of the involved skin areas by plastic procedures is the most effective means of repair.

Excision of the circumscribed nevus is the simplest method available. Wide undermining of the surrounding skin is sometimes necessary to bring about closure without undue tension. If necessary, repeated partial excisions can be undertaken. If necessary, repeated partial excisions can be undertaken. After each excision the area of involvement is reduced, aided by the natural elasticity of the skin; repeated excisions finally eradicate the deformity.[2] (Fig. I.)

This procedure cannot be applied when a large nevus is located in the vicinity of facial cavities, such as the orbit, mouth or

A   B

FIG. 2. This case is shown to demonstrate the possibility of eradication of any extensive facial skin deformity by utilizing the procedure of rotating adjoining skin flaps. This applies as well to nevi as to any type of skin involvement. A, third degree burn of right cheek, temple, upper and lower eyelids in 24 year old girl. Burn caused by fall on radiator. B, defect of cheek and temple was repaired by rotating flaps from forehead and cheek after same were delayed in order to increase their blood supply. Linear scars in photograph (taken five weeks after surgery) outline forehead, cheek and temporal flaps which were brought together following wide undermining. The partial loss of both lids was repaired by grafting of free retroauricular skin.

A   B   C

FIG. 3. A, hairy pigmented nevus in a 5-year old child, involving part of cheek and extending on side of nose and lower eyelid. Reduction in size of nevus by partial excision in horizontal and vertical diameters was done in first stage, making it possible later on to transfer a smaller forehead flap. B, shows forehead flap transferred to cheek with pedicle still attached. C, condition after return of pedicle to forehead and prior to final removal of linear scars

FIG. 4. A, pigmented atrophic scar of upper two-thirds of nasal wall in girl of sixteen, following radium treatment for nevus in early childhood. B, following excision of scar, defect repaired with a free full thickness retroauricular graft. Color of graft one year after operation, perfectly matches surrounding skin. Plastic repair done in early childhood would have eliminated a conspicuous blemish lasting for sixteen years.
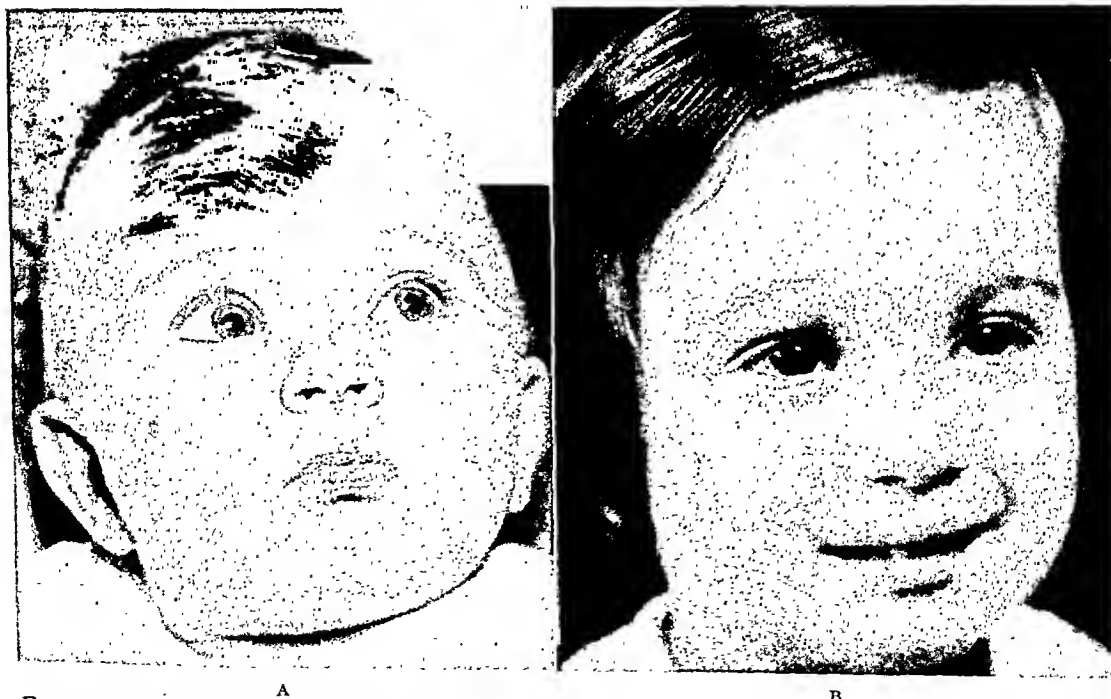
FIG. 5. A, hair nevus involving about two-thirds of forehead in six months old infant. B, condition one year later, following excision of nevus and transplantation of a free full thickness abdominal graft. Color and texture of graft were steadily improving and at present, about three years after surgery, the grafted skin matches the surrounding area. The slightly depressed scar around graft will be excised later.

nostrils. Excision in these locations, followed by closure, would necessarily bring about distortion of the part. To prevent this, rotation of the surrounding skin must be attempted. The range of possibilities provided by shifting and rotating the skin is extensive. Often, prior to rotation, if long skin flaps are to be used, it is advisable to delay the skin in a preliminary stage (by raising and resuturing in place) in order to increase the blood supply. By shifting and rotation of the delayed facial skin, we are often able to cover extensive defects of the face which seem impossible of repair without free skin grafting. (Fig. 2.)

The advantages of this procedure are self-evident: the rotated adjoining skin matches the texture and color of the face. This is not always the case even with the most successful free skin grafting, especially when large areas are involved.

The forehead provides the best matching skin for the face. (Fig. 3.) The use of forehead skin can be combined with partial repeated excisions, without causing distortion. As the forehead skin is transferred on a pedicle, this procedure entails numerous stages. Where the amount of skin required is not too extensive, selected free grafts suitable for the face are often used with success.

Free Grafts. Full consideration should be given to their texture and color. The thin dermo-epidermic grafts (Thiersch, split or razor graft) are as a rule inferior in color and texture to the full thickness grafts. They are therefore contraindicated as a rule in most facial repair except as a temporary procedure, for instance, after the removal of malignancies when final repair is delayed.

Full thickness grafts have their own special indications. The best qualified among them are those taken from the upper lids or behind the ears. (Fig. 4.)

They are the closest match to the normal facial skin. Next in order of preference are the skin grafts from the neck, inner arm and abdomen. This, of course, may vary in the individual and exceptions to the rule are frequent. In the larger skin defects it is often possible to accomplish the repair by the use of grafts from both eyelids and both ears. When scarring on the arm is to be avoided, as in young women, an abdominal free graft is given preference. (Fig. 5.)

The behavior of free grafts varies in each individual. As a rule the color and general appearance improve with time.

SUMMARY

Treatment of extensive birthmarks should be undertaken in early life.

The use of electric needle or keratolytic substances is indicated only in small raised nevi.

The use of x-rays and radium is contraindicated.

Whenever possible, a large skin blemish should be eliminated by repeated partial excisions, thereby providing a covering of the same color and texture as the surrounding area.

When complete excision, with or without rotation of the skin, is insufficient, the remainder of the blemish must be replaced by distant skin matching the face as closely as possible.

The forehead, upper eyelid, and behind the ear are the ideal sources of skin.

Thin dermo-epidermic grafts (Thiersch) as a rule do not match the face in texture and color.

REFERENCES

1. MacKee, G. M. X-rays and Radium in the Treatment of Diseases of the Skin, p. 639. Lea & Febiger. Philadelphia, 1927.
2. Maliniac, J. W. Repair of facial defects. *Arch. Surg.*, 34: 897–908 (May) 1937.

IMPURITIES IN ETHER*

H. H. BRADSHAW, M.D.

Department of Surgery, Jefferson Medical College

PHILADELPHIA, PENNSYLVANIA

THERE is a great lack of information both on the occurrence and on the effects of impurities which may occur in ether. Only since 1925 have cases of so-called ether convulsions been reported in the literature. The English have emphasized two factors which have been present in the majority of cases reported: (1) the patients have been young and (2) have had some sepsis at the time of operation.

Many theories have been advanced in an effort to explain the cause of these convulsions. These theories have been reviewed in Sears' report,[1] and also in a recent article by Lundy.[2] In the latter report, the important features of the 137 cases that have appeared in the literature have been tabulated. Lundy added four cases that had been observed by him and his associates. The mortality rate in all reported cases has been 18.9 per cent. As a result of the work done by Rosenow and Tovell, Lundy is inclined to believe that the convulsions are attributable to a neurotoxin or poison produced by streptococci in amounts insufficient to cause spasms in the absence of anesthesia, but which in the course of general anesthesia suffice to incite the muscular spasms characteristic of this condition. He also believes that the convulsions probably can be controlled by the use of barbiturates intravenously.

King[3] reported one case in which the patient died fifty-eight hours after operation. Because of the well known effects of calcium on neuromuscular irritability, calcium gluconate was given by vein. This checked the convulsions temporarily. Rovenstine[4] presented a patient who had had repeated operations with repeated convulsive seizures, but who recovered. He believes that convulsions might in some cases be due to surgical trauma, and thinks it logical to omit the term "ether convulsions," until more definite evidence convicting ether as the etiologic agent is found.

Wilson[5] reported five cases of convulsions. He believes that toxemia and hypoglycemia have a great deal to do with the production of ether convulsions.

Raab[6] observed three cases of convulsions, in two of which recovery occurred. These two received calcium gluconate by vein and the convulsions stopped within a few minutes. Raab believes that alkalosis with subsequent diminution in serum calcium is the real cause.

Woolmer and Taylor[7] reported four patients with convulsions, two of whom recovered. They believe that the heat stroke hypothesis of Dickson Wright may play a large part in the etiology of ether convulsions. The patient's temperature, the temperature of the air and the administration of atropine tend to produce an elevated internal temperature. All of their patients were treated differently, but they recommend cold sponges, the administration of carbon dioxide to accelerate heat loss and evipal by intravenous injection. In addition they use coramine, adrenalin, oxygen and artificial respiration.

Very few studies have been made on these patients. Often time is not available for making any studies, and all efforts are being concentrated on relieving the patient's condition. It is urged that whenever possible accurate clinical and laboratory studies be made and recorded, for in the presence of so many theories a few facts would be indispensable.

---

* This work was done at the Massachusetts General Hospital, Boston.

Impurities in ether have been incriminated in almost every untoward reaction that has occurred during the administration of this anesthetic. Actual proof for any of these incriminations has never been brought forward. It is only natural to blame the drug for any unusual feature which may happen, but this neither proves nor disproves anything.

A patient whom we personally observed died in convulsions during the administration of nitrous oxide-oxygen, ether anesthetic. We were fortunate in obtaining some blood for examination before the death of this patient. The findings were markedly abnormal and we are at a loss to explain them. However, a more thorough study of these patients should be made whenever possible. By this means some light may be shed on this unusual phenomenon. The case report with the findings are presented.

An 18 year old boy, suffering from obvious intra-abdominal sepsis as a result of a gunshot wound five weeks previously, was admitted from another hospital on September 28, 1934. Transfusion and intravenous fluids were given in preparation for the operation. The preoperative medication consisted of morphine sulfate gr. 1/6 and atropine sulfate gr. 1/100, given subcutaneously forty minutes before the anesthetic was started. The anesthetic was nitrous oxide-oxygen induction followed by ether. The total length of the anesthetic was forty-five minutes and the total amount of ether used was 2 ounces. There was no excitement or vomiting during the induction. Muscle twitchings of the face began with the induction, became more severe and more generalized throughout the administration, finally ending with terrific generalized convulsions and death. The respirations were fairly regular and the color pink until about ten minutes before death, at which time cyanosis began. All anesthetics were stopped and 100 per cent oxygen was given, but it had no effect. Sodium phenobarbital gr. 10 and glucose 10 per cent were given intravenously but these efforts had absolutely no effect on the convulsions.

Samples of venous blood were drawn under oil, at the height of the convulsions. These blood samples showed an oxygen content of 6.9 volumes per cent, oxygen capacity of 14.6 volumes per cent, saturation of 47.3 per cent, carbon dioxide content of 61 volumes per cent, and sugar 400 mg. per 100 cc. of blood (intravenous glucose was running into the vein). The ether was analyzed and showed aldehyde present 1:50,000 in two samples. No other impurities were found. Post-mortem examination of the body was done by the coroner but no report was obtained from him. Although 100 per cent oxygen was given for a period of ten minutes the cyanosis became more marked and the convulsions continued. The oxygen content and capacity of the venous blood were very low, with a low saturation per cent. The carbon dioxide content was within normal limits. The blood sugar figures are not reliable because of the intravenous glucose administration.

Excess carbon dioxide, hypoglycemia and lack of preoperative medication have been mentioned as causes of these convulsions. None of these conditions could have been active in this patient. The only positive finding was a low oxygen content of the venous blood despite the administration of 100 per cent oxygen. The reasons for such a state of affairs are not known. Much speculative reasoning could be offered in explanation, but this hardly seems worth while. It is known that convulsions do occur in anoxemic states.

Impurities such as acetaldehyde, peroxides, ethyl sulfide, ketones and alcohol are known to occur in ether. The first three impurities mentioned are said to be toxic if present to the extent of 0.5 per cent or more.[9,10] There is apparently no uniformity of opinion regarding the rate of formation of impurities after a can of ether has been opened. The disposition of partly used cans is always a matter of some doubt. Also when oxygen has been allowed to pass through ether as is done in modern anesthetic machines, the possibility of oxidation with the formation of impurities arises.

With the above facts in mind, we have made some observations on the purity of ether, a product of one of the standard manufacturing companies. Ether was placed in copper containers, in clear glass

bottles and in 5 pound cans as dispensed by the manufacturer. The copper containers were equipped with copper screw caps. The other containers were stoppered with ordinary corks. Each of these containers was thoroughly shaken and a sample of ether pipetted and tested once a week for twelve months. The tests used were as follows:

1. Aldehyde. Nessler's reagent. Ether having aldehyde present 1 part in 80,000 gives a positive reaction.

2. Peroxide. 10 per cent solution of cadmium potassium iodide. Ether having $\frac{5}{100}$ of 1 per cent present gives a positive test.

3. Acidity. The sample is evaporated at room temperature. Distilled water is added to the residue. Light blue litmus paper is changed if acid is present.

The results of these tests are shown in Table 1.

Further tests for impurities in ether were carried out in the following manner: Oxygen was passed through ether in an anesthetic machine at varying rates of flow from 200 c.c. to 500 c.c. per minute for a period of five hours. All tests were negative for impurities on repeated examinations. This procedure was modified so that instead of immediate sampling, the ether was allowed to stand in the anesthetic machine overnight and sampled the following morning. This test was repeated again and again with completely negative results.

CONCLUSIONS

1. A case of fatal convulsions, occurring during nitrous-oxide-oxygen-ether anesthesia is reported.

TABLE I

TEST FOR IMPURITIES IN ETHER

Number	Container	Tests Made	Aldehyde	Peroxide	Acid	Odor
1	Copper	56	Negative	Negative	Negative	Negative
2	Copper	56	Negative	Negative	Negative	Negative
3	Ether can	56	Negative	Negative	Negative	Negative
4	Ether can	56	Negative	Negative	Negative	Negative
5*	Clear glass bottle	56	Positive on 42nd test	Negative	Negative	Negative
6	Clear glass bottle	56	Negative	Negative	Negative	Negative

* After container No. 5 had once become positive, the aldehyde content increased perceptibly during the remaining test periods.

4. Odor. The sample is allowed to evaporate on a filter paper. Impurities give a perceptible foreign odor as the last portions disappear from the paper.

Further tests were carried out to determine the sensitivity of the methods used. Known solution of impurities in ether were made up as follows: (1) aldehyde, 1 part in 80,000; (2) peroxide, 1 part in 5,000. When the U.S.P. test for aldehyde was used, no reaction was visible in two hours. When Nessler's reagent was used, an immediate yellow color with a heavy sediment occurred within a few minutes. Using the cadmium potassium iodide test for peroxide an immediate yellow color occurred.

2. Repeated tests for impurities in ether from various types of containers, demonstrates excellent stability. Only one sample showed a positive test, and that after ten months.

3. The passage of oxygen through ether did not produce any impurities, during the period studied.

REFERENCES

1. SEARS, J. B. Later ether convulsions; report of 2 cases. *J. A. M. A.*, 100: 1150, 1933.
2. LUNDY, J. S. Convulsions associated with general anesthesia. *Surgery*, 1: 666, 1937.
3. KING, H. J. Convulsions under ether anesthesia. *Am. J. Surg.*, 30: 183, 1935.
4. ROVENSTINE, E. A. Convulsions during ether anesthesia: a case report. *Anesth. & Analg.*, 14: 40, 1935.

5. WILSON, W. S. Convulsions under ether anesthesia. *Anesth. & Analg.*, 14: 281, 1935.
6. RAAB, A. Convulsions during ether anesthesia. *Anesth. & Analg.*, 15: 295, 1936.
7. WOOLMER, R. F., and TAYLOR, S. Late ether convulsions; a study based on four cases. *Lancet*, 1: 1005, 1936.
8. WILSON, A. C. R. Ether convulsions. *Lancet*, 1: 1117, 1927.
9. BOURNE, W. The effects of acetaldehyde, ether peroxide, ethyl mercaptan, ethyl sulphid and several ketones—di-methyl, ethyl methyl and di-ethyl, when added to anesthetic ether. *Anesth. & Analg.*, 6: 131, 1927.
10. MENDENHALL, W. L., and CONNOLLY, R. Pharmacological effect of impurities in ether. *J. Pharm. & Exper. Therap.*, 43: 315, 1931.

THE accurate figure of incomplete cures or recurrences [after subtotal thyroidectomy] is difficult to establish, but an approximation of the truth is obtained by Lahey's statement, who found in a follow-up study that, five years after operation, exophthalmos of considerable degree remained in 21.0 per cent of the cases.

From—"The Endocrine Glands" by Max A. Goldzieher (Appleton-Century).

PHYSIOLOGIC ABDOMINAL INCISIONS

H. P. SLOAN, M.D.

The Sloan Clinic

BLOOMINGTON, ILLINOIS

IT is not the closing of an incision which causes adhesions, herniae, and evisceration; it is the opening. Regardless of whether one uses silk, wire, or gut, one cannot repair damage which has been too great. There is no way by which one can destroy body tissues, replace them immediately with mechanical substitutes and expect those tissues to function. Those who look to these mechanical aids in closing incisions are looking only at the result. If they would look at the cause they would then be able to set about constructively seeking a real method of eliminating postoperative wound ruptures. The cause of ineffective closure is obvious. It is the incision.

White[24] says: "In the symposium mentioned, and the discussion which followed, stress was placed upon the suture material, the method of closing the wound; reference was made to infection, the cachectic condition of the patient, the pulmonary complications and other indeterminate factors, but very little was said of the type of the incision and the disturbance of the innervation of the muscles involved in the hernia." Our own experience has convinced us of the importance of maintaining nerve supply. The blood supply is of lesser importance because of the greater power of regeneration of the circulatory system.

Cave[3] says: "It seems reasonable to presume that though considerable time and concentration are utilized in the performance of the operation within the abdomen, often a two hurried and careless closure is effected by a weary operator or an inexperienced assistant." Cave found that in eleven years at Roosevelt Hospital 6.02 per cent of all herniae were incisional, 2.2 per cent of incisional herniae occurring in 5,366 abdominal operations. Cave reports 444 patients operated on through a McBurney incision for acute appendicitis. In 282 cases not drained no incisional hernia occurred. In the remaining 162 cases, which were drained, twenty-one incisional herniae developed. Obviously these herniae are attributable to the fact that drains were used, not to the incision.

Fallis[7] has published a report on fifty eviscerations from a series of 7,903 consecutive laparotomies. In considering forty-nine of these cases he finds that twenty-six ruptures occurred in upper abdominal incisions. It is interesting to note that all of the incisional ruptures occurred in the use of various unphysiologic incisions while none occurred with the use of the Pfannenstiel or McBurney incisions, the only two physiologic incisions listed. While Fallis draws attention to the fact that no ruptures occurred after the use of the McBurney, he dismisses the Pfannenstiel as "infrequently used."

Bettman and Lichtenstein[1] reported thirty-two cases of evisceration in 1936. In compiling their figures they eliminated 5,000 appendectomies which were done through a McBurney incision because "many authors felt it is not conducive to evisceration." They also state, "We did not feel that the method of wound closure made a great deal of difference for almost as many types of sutures and methods of closure are used in the Michael Reese Hospital as there are surgeons working."

Heineck[9] has said, "The abdominal wall acts as a buttress for the abdominal viscera. It assists in regulation of intra-abdominal pressure; it aids in processes of respiration, urination, defecation and parturition. All incisions of the abdominal

wall weaken it, impair its integrity and predispose to hernia formation." While Heineck does call attention to the fact that incisions should be no longer than necessary, he stresses the prevention of incisional herniae by minimum traumatism, perfect hemostasis and asepsis, the use of absorbable suture material, careful closure and minimum drainage—he makes no comment upon the use of physiologically correct incisions except the McBurney.

Starr and Nason[23] of Boston give 0.61 per cent of definite evisceration in 2,455 abdominal operations. On none of these patients was a physiologically correct incision used.

Seventy-five per cent of the cases of evisceration reported in a survey to McCauliff[14] occurred after a midrectus incision. Transrectus and midline incisions accounted for most of the remaining 25 per cent. Nearly all occurred above the umbilicus. No occurrence was recorded in any report where muscle sliding or muscle retracting incisions were used.

Shipley[19] asks, "What can be done to lessen the percentage of broken down infected abdominal incisions?" Speaking about abdominal operations he says, "The final step is one about which there is lack of agreement, much anxiety, and no little grief—the method of closure. Many different materials are used. No single way of suturing the wound edges has been proved satisfactory. The problem calls urgently for solution."

There is a striking incompleteness about most of the papers on this subject. Classification is confused. Postoperative wound rupture is a term constantly used. It may mean anything from a simple drain hernia to complete evisceration. Many figures have been compiled showing age incidence, seasonal effects, preponderance of sex and other factors. Many times mention of the type and length of incision used is omitted; generally the remote results are disregarded. It is not reasonable to evaluate the detrimental results of unphysiologic incisions in terms of evisceration alone. Figures from a number of reports indicate the prevalence of evisceration to be about 0.6 per cent and the mortality among these cases is from 35 to 70 per cent.

The more remote results are the postoperative herniae (those in which evisceration does not occur). The incisional hernia occurs up to twenty years after operation, but the responsibility must still be laid on the original incision. Masson[16] reviewed 5,502 cases of hernia treated at the Mayo Clinic and found 10.8 per cent of them to be incisional. I have seen too many patients whose original gall-bladder operation occasioned as many as four subsequent operations in an effort to repair the incisional hernia. The total number of patients who have a postoperative incisional hernia cannot be determined one month, six months, or even six years after operation. While most of these herniae appear soon after operation, an appreciable number occur many years following. Particularly is this true in those cases in which drains are not left. In our experience over 85 per cent of the incisional herniae which follow vertical incisions without drainage occur during the sixth to the tenth year following operation. In 128 patients of ours[20] with no external evidence of hernia five or more years after operation through a vertical incision, only nine were found to have a satisfactory union of the posterior sheath.

The high incidence of postoperative adhesions to the inner side of the scar is of great importance. In a series of 367 of our cases operated upon five or more years previously through vertical incisions there were only five who did not have adhesions to the under side of the scar. Some factor other than imperfect closure must be responsible for this high incidence, for many patients have strong, apparently perfect scars, but with massive adhesions under them.

No amount of research on suture materials, duration of operation, age or sex incidence, type or method of suturing, or

method of drainage, is going to eliminate the results of poor incisions. All of these things have importance but the primary

Fig. 1.

factor must be eliminated first. While we know of no way to eliminate the unsatisfactory results by an easier procedure, we can eliminate a very high percentage of these poor results by using physiologic incisions. True, until one is accustomed to their use they seem more difficult. The technique is more complicated and a little more time is required. The marked decrease in the amount of postoperative care and shock offsets the additional time used and the satisfaction of better postoperative results well pays both the surgeon and the patient for the additional time and labor spent.

In deciding which incision to use the surgeon realizes that three requirements should be met, or approached as nearly as possible: the incision should be physiologically correct; it should provide a good operative field; and, the result should be a minimum of disfigurement. Of course, sometimes all of these desirable effects cannot be obtained. Cosmetic result frequently must be sacrificed. However, the only time the physiologic incision should be sacrificed is where speed is essential to save the patient.

With these requirements in mind let us look at the most widely used abdominal incisions. In Figure 1 the various incisions have been reproduced on only one side

of the body although most of them are applicable to both sides.

If we are considering these incisions
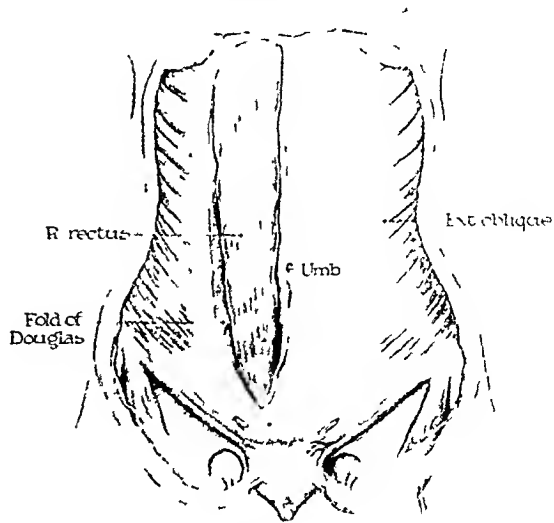
Fig. 2.

with their physiologic correctness in mind we are not particularly concerned with the skin incision except that any incision should be made along the "fissure lines of Langor."

The most important structures encountered in abdominal surgery are the three aponeurotic transverse muscles, the external and internal oblique and the transversus abdominis. The fibers of the aponeuroses of these muscles vary both ways, from the horizontal to 30 degrees. These aponeuroses fuse and form the anterior and posterior sheaths of the recti and in the midline the linea alba. The outermost of these muscles, the external oblique, after its origin from the lateral surfaces of the fifth to the twelfth ribs, becomes a thin flat muscle whose fibers run downward, forward and inward. The posterior portion is attached to the anterior half of the outer lip of the crest of the iliac bone. The balance of the muscle has a broad aponeurosis along its entire medial edge, forming part of the anterior sheath of the rectus. Along an imaginary line drawn between the anterior superior iliac spine and the umbilicus, an aponeurosis runs downward, inward and forward, extending between the anterior superior

iliac spine and the spine and crest of the pubic bone to form the inguinal or Poupart's ligament.

FIG. 3.

The internal oblique, thin and fan-shaped, arises from the lumbodorsal fascia, the anterior two-thirds of the intermediate lip of the iliac crest, and from the lateral half of Poupart's ligament. The fibers run in different directions, forming a wide fan. The posterior fibers run upward and forward to attach to the tenth, eleventh, and twelfth ribs. The remaining fibers extend to the lateral border of the rectus muscle forming an aponeurosis which immediately splits into two layers or lamellae, the anterior of which fuses with the aponeurosis of the external oblique muscle to form the anterior sheath of the rectus. The posterior lamella fuses with the aponeurosis of the transversus abdominis muscle to form the posterior sheath of the rectus.

The transversus abdominis muscle arises: from the posterior surfaces of ribs seven to twelve by six slips which interdigitate with the slips of origin of the diaphragm; from the lumbodorsal fascia; from the anterior two-thirds of the internal lip of the iliac crest; and, from the lateral third of the inguinal ligaments. In the main the fibers run horizontally, per-

pendicular to the midline. The muscle ends in an aponeurosis which helps the upper three-fourths of the posterior and
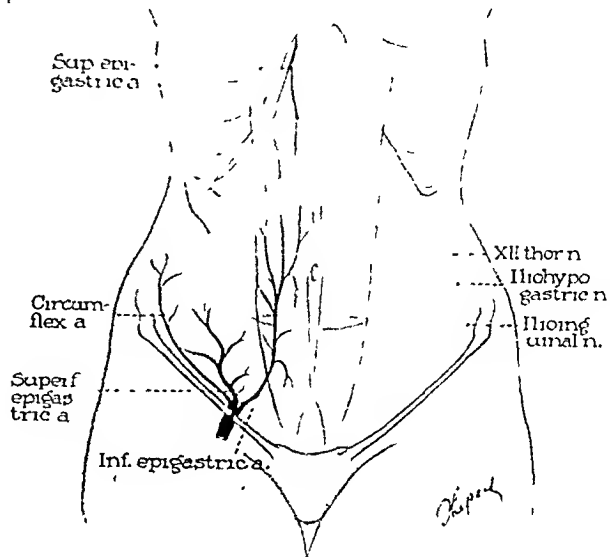
FIG. 4.

the lower fourth of the anterior sheaths of the rectus.

The most important surgical consideration in connection with these muscles is the fact that midway between the umbilicus and pubic bone downward the posterior sheath of the rectus is deficient. From this linea semicircularis (semilunar fold of Douglas) downward the posterior lamella of the aponeurosis of the internal oblique and the aponeurosis of the transversus abdominis fuse with the anterior lamella of the internal oblique and the aponeurosis of the external oblique to form the *anterior* sheath of the rectus.

The blood supply of the anterior abdominal wall is derived principally from the superior and inferior epigastric arteries which run the same direction as the recti fibers, and the intercostal arteries which run perpendicular to the recti fibers. All nerves run perpendicular to the recti fibers. The nerve supply is derived from the seventh to twelfth thoracic nerves, from the iliohypogastric and ilio-inguinal branches of the first lumbar nerve. No matter what incision is used the blood

supply is sufficient to compensate cut blood vessels. Nerve damage inflicted by a longitudinal incision, however, cannot

FIG. 5. Skin incision from the two anterior superior spines of the ilium downward in a curve through a point two fingerbreadths above the pubic bone. The flap is pulled up and the anterior sheath exposed.

be compensated and any muscle or aponeurosis medial to the incision will atrophy. Transverse incision of the abdomen preserves the innervation. Lack of nerve supply and cutting the aponeuroses crosswise of the direction of its fibers are the causes of ventral hernia—and never loss of blood supply. In the midline incision postoperative hernia is due, not to atrophy of muscle, but to cutting the aponeurotic fibers of all transverse muscles perpendicular to the direction of their fibers. Also, the blood supply coming from terminal endings is deficient in the linea alba.

There are six consequences resulting from poor incisions: (1) hernia (which may occur many years after operation) and adhesions; (2) difficulty in closing abdomen; (3) postoperative pneumonia due to tension making the patient afraid to cough; (4) longer confinement in bed; (5) poor cosmetic scar; (6) evisceration under increased abdominal pressure. Which tissues in the abdominal wall are physiologically the most important from the viewpoint of prevention of adhesions or hernia? Adhesions are caused by imperfect closure of the peritoneum or by failure

of the closure to hold. While it is rupture of the peritoneum which causes the adhesions, it is the lack of restraining pressure

FIG. 6. The anterior sheath of the rectus is split transversely and retracted, exposing the rectus and pyramidalis muscles when found. Wide variation is found in regard to the pyramidalis. Blunt separation and retraction of the recti permits the incision of the fascia and peritoneum longitudinally.

on the part of the aponeurosis and fascia forming the posterior sheath of the rectus which enables the peritoneum to open. We may conclude, then, that the most important tissue of the abdominal wall, physiologically, is the aponeurosis forming the posterior sheath, and the transversalis fascia. Following are, in the order of their importance, the peritoneum, the rectus muscle, the anterior sheaths of the rectus and the deep and superficial fascia.

In the upper abdomen the transversalis fascia is a thin layer surrounding the entire abdominal cavity although known by different names in different areas. The layer is elastic and its fibers are circular and always perpendicular to the longitudinal axis of the body. As far as the surgeon is concerned it may be considered as one layer with the peritoneum in the lower abdomen and with the peritoneum and posterior sheath in the upper abdomen and is treated as such. For this reason the posterior aponeurosis and the fascia are the main hernia preventing factors in upper abdominal work while the

anterior aponeurosis is the restraining unit in the lower one-fourth.

If we keep in mind the anatomy of the

FIG. 7. An incision is made through the skin and fat down to the fascia, from the ensiform process to a point 3 cm. above the umbilicus and continued outward and downward on each side of the umbilicus to a point 4 cm. below the umbilicus on either side, leaving a V-shaped piece of skin and fat around the umbilicus.

abdominal wall and the direction of the fibers in its tissues, it is evident that no longitudinal incision through this fascia can be physiologic. If we recognize the importance of cutting as little as possible of the nerve and blood supplies, and not cutting aponeuroses crosswise, we leave as truly physiologic incisions only those which involve muscle-splitting.

It is, of course, impossible to perform all surgery through muscle-splitting incisions. Our efforts must be bent toward the nearest approach to this ideal, at the same time retaining the maximum degree of utility from the standpoint of operative field, speed of operation and accessibility.

In a short paper it is impossible to discuss the many incisions which have been advocated for abdominal work. The surgeon who desires further information about any of these incisions may find a most complete chapter on them in Spivack's book.[22]

The difference, externally, between the physiologic and unphysiologic incision is apparent long after their use. The unphysiologic incision often produces a broad scar which very evidently is the result of any undue strain placed upon it. In our experience we have usually found upon reoperation that these broad scars from unphysiologic incisions have adhesions under them which have resulted from an improperly closed peritoneum or the giving away of sutures in the innermost layers before union has taken place.

Over 90 per cent of all surgery in our clinic is done through one of three incisions: a modified McBurney, the Pfannenstiel, and the Sloan. These incisions are nearly perfect physiologically and produce the best results, we believe.

The advantage of the McBurney "gridiron" incision is that it is more nearly perfect, physiologically, than any other. Placing the incision just inside the iliac spine permits direct access to the mesoappendix without displacing loops of the small intestine mesially or rotating the head of the cecum. While some surgeons do not use it, claiming that it is slower and that it is difficult to remove an appendix which is tied down by adhesions retrocecally, the surgeon accustomed to its use finds no difficulty in severing the tied-off appendix at the colon and then bringing it out proximal end first. This is simplified by the modified incision. It is, perhaps, a slower procedure but well worth the additional time, being paid for by the rapid recovery and the certainty of no postoperative hernia. Most important in the case of rupture, the abscess is walled off by the omentum into the lower right quadrant, facilitating drainage while leaving the infection localized. The only possible disadvantage of this incision is that the appendiceal region and that of

the right ovary are the only ones which may be reached. It is our practice upon entering the abdomen with the McBurney

roses of the transversus abdominis and internal oblique, in the lower quarter of the abdomen, are part of the anterior
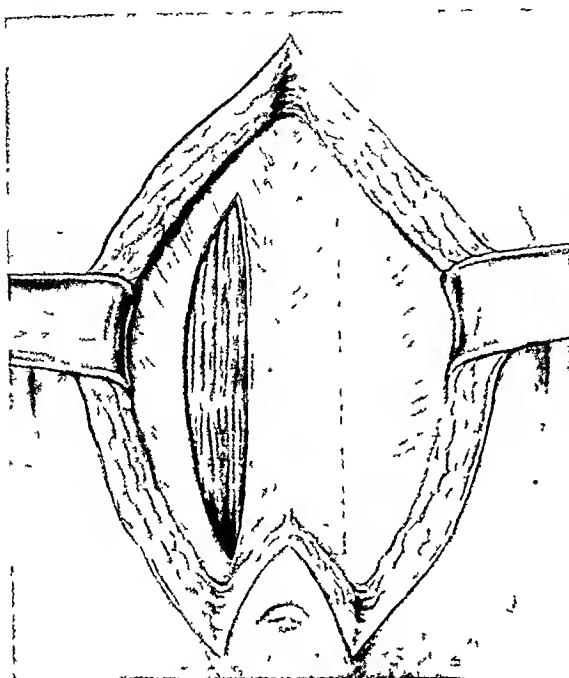
FIG. 8. After palpation of the edge of the rectus by the finger, vertical incisions are made through the anterior sheaths of the recti about 1 cm. distal from the mesial edge of the recti. Upon the length of these two incisions will depend the amount of exposure that it is possible to obtain.
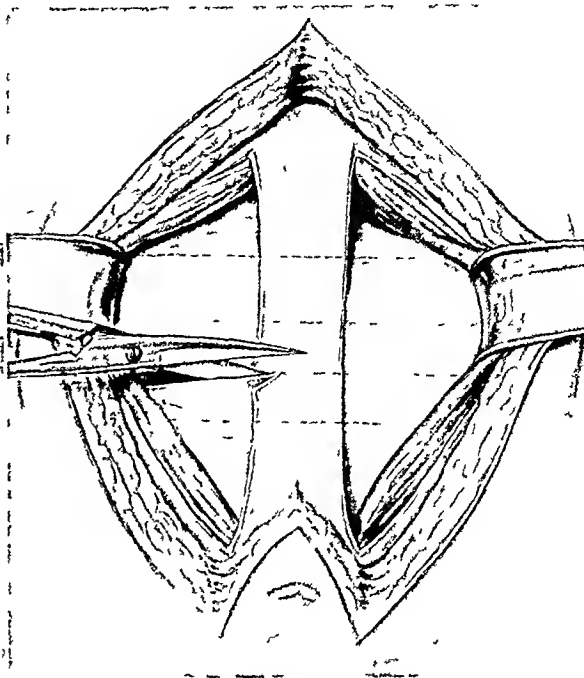
FIG. 9. After retractors are in place and the posterior sheath of the rectus exposed a transverse incision is made through the exposed posterior reflexion of the sheath of the rectus and the peritoneum. Dotted lines indicate locations which may be chosen.

and discovering the need for more extensive surgery or additional room, to extend the skin incision downward laterally, joining the McBurney to a Pfannenstiel. By doing this we are enabled, when necessary or advisable and without any detrimental effect on the patient, to do any additional work necessary in the lower abdomen and still maintain a physiologic incision. If preferred, the Fowler or Weir extension incision can be used. In this incision the rectus is retracted medially and the anterior sheath, peritoneum and transversalis fascia are incised transversely to the midline from the lower end of the McBurney.

For all lower abdominal surgery (except those cases of large tumors or extensive exploratory) the Pfannenstiel[18] incision offers a practical and nearly physiologic solution. If we remember that the aponeu-

sheath, then we can realize that the longitudinal incision through fascia and peritoneum is not so serious as long as we still make the transverse incision (parallel to the fibers) through the aponeurosis which is the anterior sheath.

The Pfannenstiel incision is an old one which fell into disuse. It was one of the earliest forerunners of physiologic incisions. The incidence of postoperative hernia and adhesions is almost negligible; its application is wide and affords ample field for ordinary surgery. The scar is not disfiguring and the closure is easy.

The Pfannenstiel has several disadvantages. It cannot be extended sufficiently for removal of unusually large tumors; however, these are usually located by palpation and in those instances another incision used. It is harder to work through the Pfannenstiel than some other incisions and the technique is more extensive. In

our experience these disadvantages are far outweighed by the superior result obtained.
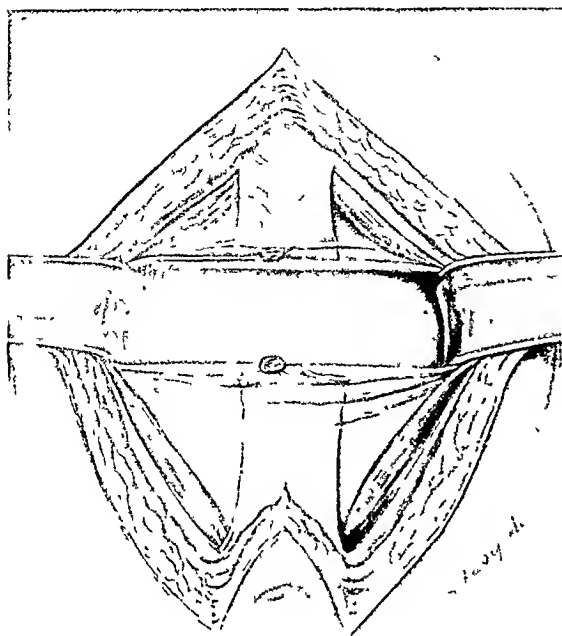
FIG. 10. The incision is extended across the linea alba parallel to the direction of the fibers of the aponeuroses from the outer edge of one rectus muscle to the outer edge of the other.

The application of the Pfannenstiel incision is shown in Figures 5 and 6. The skin is incised from the two anterior-superior spines of the ilium downward in a curve through a point two finger-breadths above the public bone. The flap is pulled up and the anterior sheath is exposed. The anterior sheath of the rectus is split transversely and retracted, exposing the rectus and pyramidalis muscles when found. Wide variation is found in regard to the pyramidalis; sometimes it is particularly large and well developed, at other times it is entirely missing. Blunt separation and retraction of the recti permits the incision of the fascia and peritoneum longitudinally.

In the event that this opening is too small for removal of the uterus or large tumors a vertical incision of the anterior sheath may be extended from the lowest point of its arch downward almost to the symphysis pubis. This permits two small triangle flaps to fold back. The anterior sheath can be further retracted as may the recti, and the incision through the transversalis fascia and peritoneum is extended downward. If the pyramidalis muscles are present and at all well developed the vertical extension must be extended along the lateral edge of one of them and the pyramidalis retracted on one side along with the rectus on that side.

The question has been brought up that the extension of the Pfannenstiel downward to the symphysis pubis is unphysiologic. This is quite true. However, this small unphysiologic part of the incision is at the lower apex of all the abdominal muscles at a point where the distance of extension and flexion from intra-abdominal pressure is slight. We have never found an incisional hernia following this procedure unless a drain is used.

The Sloan[21] incision is a modification of the old Winkleman incision which was used in Europe many years ago. Since it was first shown in 1927 a number of men in this country have started using it. These have quickly extended its use. To others it has seemed a formidable process with a very difficult technique. In our own experience, however, this incision has been extremely successful both surgically and postoperatively. Its technique is completely illustrated and explained in Figures 7 to 18 inclusive.

This is a fast, simple and adequate incision for most work. Only one opening need be made in the anterior sheath; the posterior sheath, fascia and peritoneum are opened transversely by blunt dissection. At the same time, should it prove desirable, the skin opening may be extended on the other side, another incision made in the anterior sheath and the linea alba cut transversely, forming the full incision. (Fig. 19.)

Spivack[22] advances as the only objection to the Sloan incision, the fact that the anterior sheath of the rectus is cut longitudinally. This fact prevents the incision
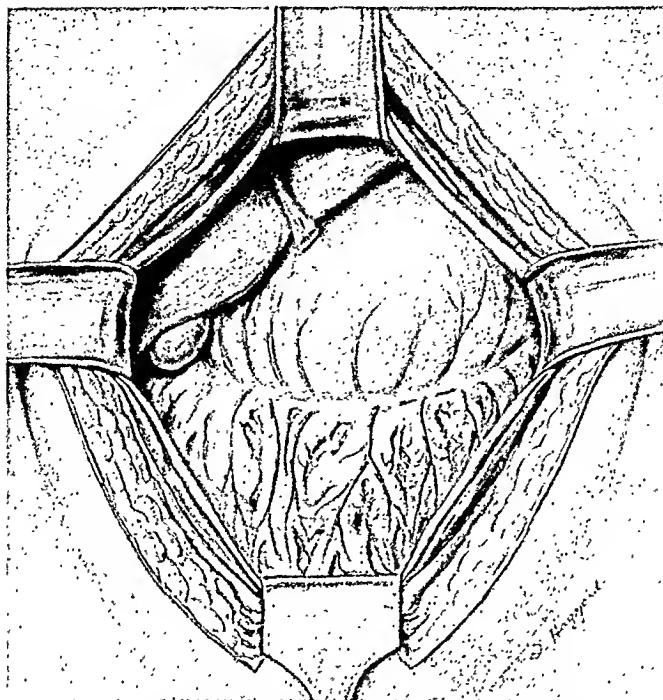
FIG. 11. Retraction upward and downward affords an opening with a diameter about equal to the length of the incisions in the anterior sheaths of the rectus muscles.
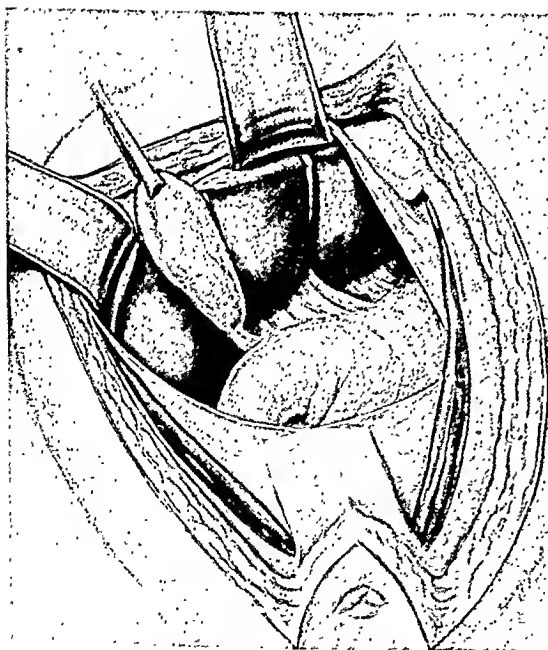
FIG. 12. Retraction upward and outward affords access to the gall-bladder region. The opening can be retracted over the regions of the spleen or appendix also.
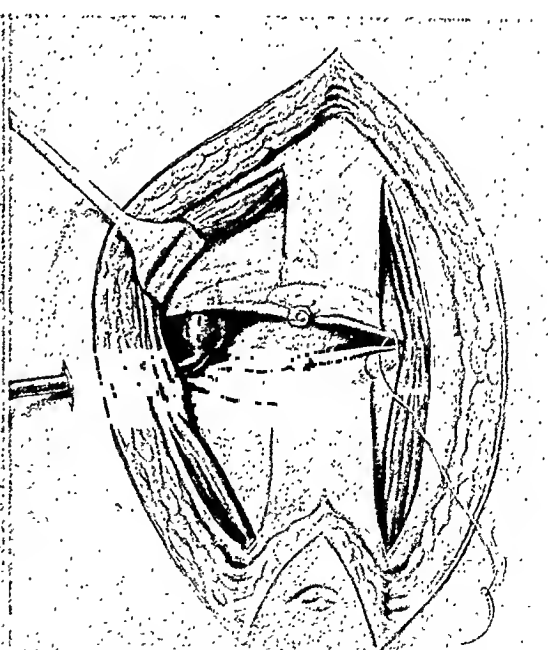
FIG. 13. In cholecystotomy the drainage tube is brought out through a puncture wound and the gall-bladder is brought to the abdominal wall at any point desired.
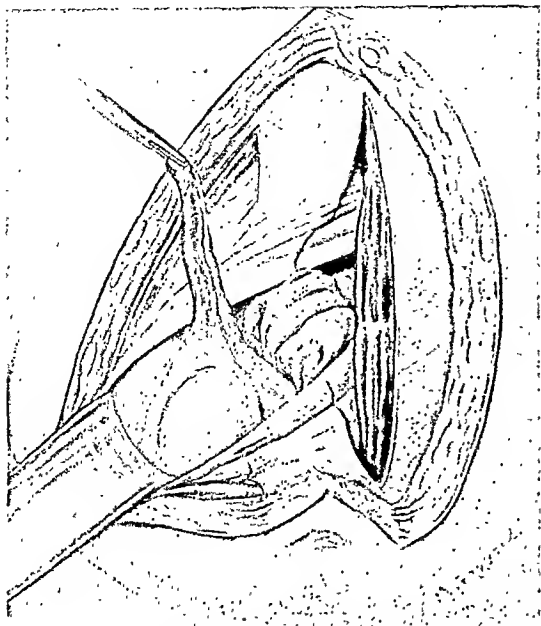
FIG. 14. The appendix is more readily dealt with through this incision than through a high right rectus incision of any reasonable length.
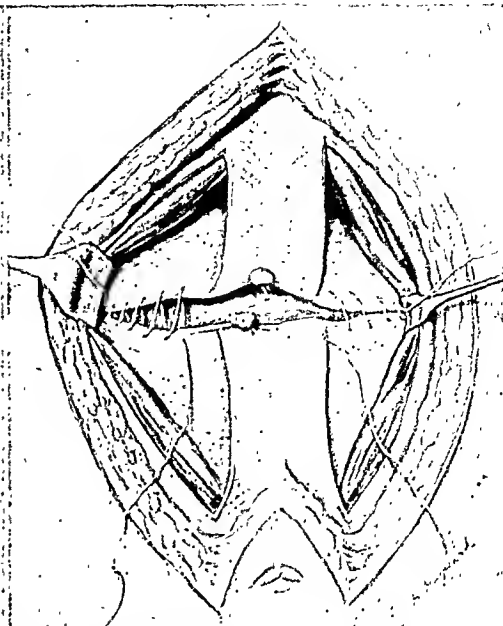
FIG. 15. The closure of the peritoneum and posterior layer of aponeurosis is begun at the outer ends of the transverse incision.
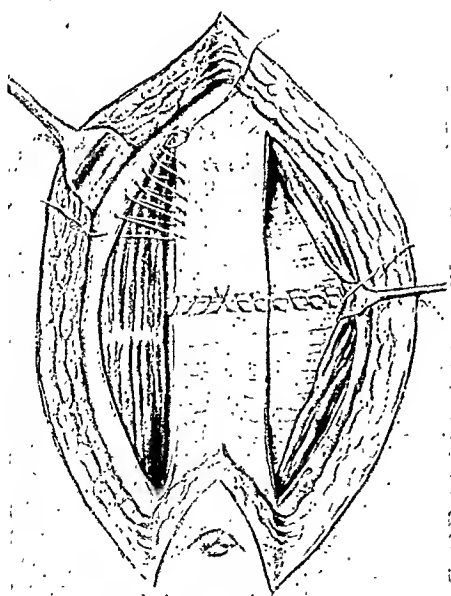
FIG. 16. No difficulty is experienced in closing the abdomen even with the largest and fleshiest patient as no tension whatever is required to bring the edges together.
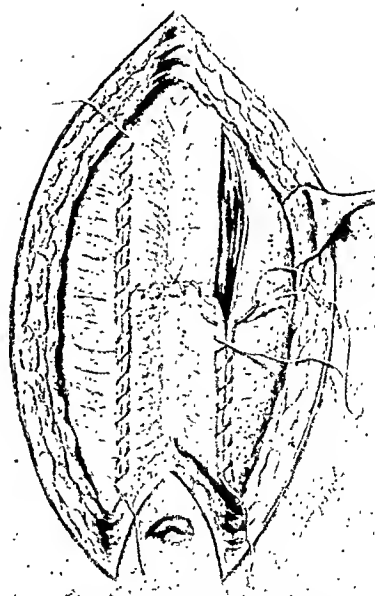
FIG. 17. No appreciable tension upon the sutures required to close the anterior sheath.

from being perfect physiologically, but we must remember that the anterior sheath bears very little strain. If the posterior sheath is intact, there is practically no strain upon the sutures in closing the anterior sheath and skin.

Very few nerves and blood vessels are cut. We have never found atrophy of the rectus medial to the incision in later reopening this incision. The incidence of pneumonia is so rare that I cannot remember one case. The patient is more comfortable by the mere fact that he has less pain. He breathes much more deeply with a resulting decrease in postoperative respiratory complications.

A not inconsiderable advantage of this incision is that there are no sutures required to hold the enormous intra-abdominal pressure. We never use any form of "through-and-through" suture. Healing is complete and there is no possibility of eviscerating. We have had

entrance and closure are made. In the unphysiologic incision the aponeurosis fibers snap back like a taut rubber band
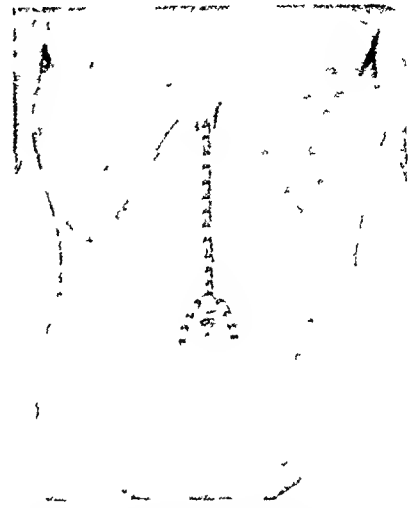
Fig. 18. The skin and fat flaps fall together with surprising readiness. In fact, skin clips are all that are required to hold the flaps of skin and fat together, and the scar is a fine line much superior to that of the usual incisions.
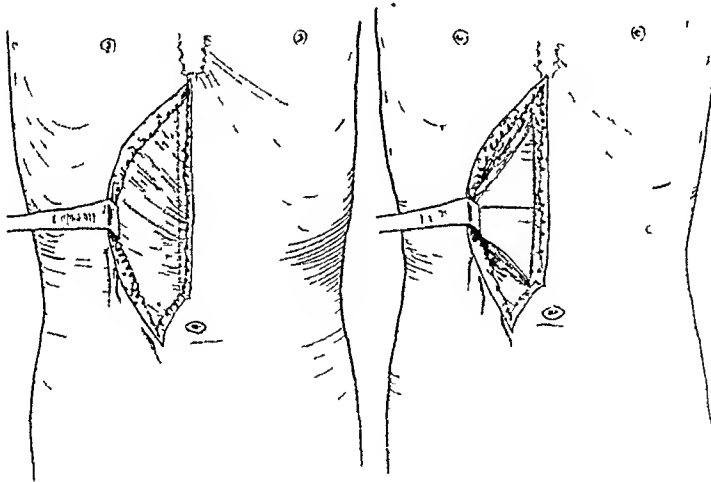
Fig. 19. Keeping in mind the full incision, it is easy to see that in a majority of cases the incision need only be made on one side. Even this unilateral incision has a large advantage over the Winkleman for it may be retracted to reach the appendix and affords a larger and more accessible field of operation. We seldom need to use both sides but often extend the right side incision across the linea alba to the edge of the left rectus muscle.

patients get out of bed during delirium with no ill effects. Comparative proof of all these statements is readily available if upper abdominal surgery is done each way and comparison noted in the way

when severed in distended cases. A great deal of strength is needed to bring them together and it is a question as to whether the sutures will hold. Even when they apparently do hold the resulting scar

shows the result of stretching. Nearly always there are adhesions under it and the question is always present as to whether there is a partial hernia and whether the incision will not entirely reopen. In the Sloan incision there is no cutting of musculature under tension, and in closing, the muscles and flaps practically fall together. The skin incision is closed with clips and the resulting scar is a thin line.

Postoperative care is much easier than with the usual incisions. There is no need of silkworm retaining sutures which we use with any other upper abdominal incision. No other upper abdominal incision approaches the perfect physiologic incision so closely and is at the same time so wide in its application.

CONCLUSIONS

Conclusions may be made upon the basis of available material, although it is to be regretted that no figures can be found to bear out the true picture. It is my definite conviction that 15 to 20 per cent is a conservative estimate of incisional failures in operations performed through vertical abdominal incisions. That the incidence is high is proved by the fact that no complete figures have been published upon it.

A survey of a reasonable number of patients operated upon through unphysiologic incisions would be invaluable if:

1. Complete information about the length and location of the incision were given. (It is recognized that a small vertical incision is not nearly so precarious as a longer one, the amount of abdominal strain increasing as the *square* of the length.)

2. Complete information were given as to the extent of the hernia, indicating whether evisceration occurred, whether the closure through the musculature failed to hold, or whether the hernia was occasioned by drainage.

3. The series were carried through a minimum of twelve (preferably fifteen) years.

4. The presence of, and the complications from, adhesions were considered.

5. Some way could be devised to test incision hernia for our records even though it is not bad enough to need re-operation.

We do know:

1. That evisceration occurs in approximately ½ to 1 per cent of all unphysiologic abdominal incisions.

2. Figures published upon postoperative hernia admit 2.2 per cent to 8.9 per cent (from all references). These figures are based upon five years or less and can therefore be accepted as minimal. It would be absurd to suppose that only 2.2 per cent of these incisions produce hernia and .5 per cent (one out of every four) eviscerate. These figures are, of course, compiled about those patients who are reoperated or treated by the original surgeon. It is not known how many from these series go to other surgeons in less or more than five years for repair. Neither is it known how many retain unrepaired herniae.

3. That no figures are available regarding the incidence of adhesions. In 367 cases of postoperative hernia, treated by our clinic, all but four being over five years past original operation, only five had no adhesions. It is only reasonable to estimate a greater number of postoperative adhesions than postoperative herniae. If there is hernia there are adhesions (in 98.6 per cent) and there may be adhesions without hernia.

4. That by using physiologic incisions we eliminate those cases whose mortality is highest, the cases of evisceration. (Mortality is variously placed from 35 to 70 per cent.)

5. That by the same means we can practically eliminate all of the incisional herniae except those which have been caused by drains. These herniae are usually easily repaired under local anesthesia.

6. That in accomplishing the enormous reduction in incidence of hernia we reduce the occurrence of adhesions.

7. In doing all of these things we promote a greater number of patients to a better postoperative condition and reduce mortality. In the process the patient recovers more quickly, endures less pain and discomfort, and makes a comparative financial saving.

We also know that we work longer and harder in the performance of our surgery. Our technique is more tedious and the operative field sometimes confined. Often it is necessary to make two good incisions where one poor one would do. We think it is worth doing.

REFERENCES

1. Bettman, R. B., and Lichtenstein, G. M. Evisceration following abdominal operations. *Arch. Surg.*, 32: 721, 1936.
2. Boechmann. *St. Paul M. J.*, June, 1910.
3. Cave, H. W. Incidence and prevention of incisional herniae. *J. A. M. A.*, 101: 2038, 1933.
4. Coley, B. L. Three thousand consecutive herniotomies, with special reference to recurrence, based on 837 followed cases. *Ann. Surg.*, 80: 242, 1924.
5. Colp, R. Disruption of abdominal wounds. *Ann. Surg.*, 99: 14, 1934.
6. Eliason, E. L., and McLaughlin, C. Postoperative wound complications. *Ann. Surg.*, 100: 1159, 1934.
7. Fallis, L. Postoperative wound separation: review of cases. *Surgery*, 1: 523, 1937.
8. Glenn, F., and Moore, S. W. The disruption of abdominal wounds. *Surg. Gynec. & Obst.*, 65: 16, 1937.
9. Heineck, A. P. Postoperative or incisional herniae of the anterior and lateral abdominal wall. *Illinois M. J.*, 67: 529, 1935.
10. Hinton, J. W. Allergy as an explanation of dehiscence of a wound and incisional hernia. *Arch. Surg.*, 33: 197, 1936.
11. Jenkins, H. P. Clinical study of catgut in relation to abdominal wound disruption. *Surg., Gynec. & Obst.*, 64: 648, 1937.
12. Keith, H. Closure of abdominal incisions. *Brit. M. J.*, 2: 550, 1927.
13. Lahey, F. H. Management of some complications following abdominal operations. *J. A. M. A.*, 89: 1735, 1927.
14. McCauliff, G. T. Rupture of wounds following abdominal operations. *Iowa State M. J.*, July, 1933.
15. McNealy, R. W., and Lichtenstein, M. E. Postoperative hernia. *Am. J. Surg.*, 18: 90, 1932.
16. Masson, J. C. Postoperative ventral hernia. *Surg., Gynec. & Obst.*, 37: 14, 1923.
17. Meleny, F. L., and Howes, E. L. Disruption of abdominal wounds with the protrusion of viscera. *Ann. Surg.*, 99: 1, 1934.
18. Pfannenstiel. *Somul. klin. Vortr. Cynak.*, No. 97, 268, 1900.
19. Shipley, A. M. Disruption of abdominal wounds: an unsolved problem. *Surgery*, 1: 517, 1937.
20. Sloan, E. P. Abdominal incisions, *Am. J. Obst. & Gynec.*, 23: 226, 1932.
21. Sloan, G. A. New upper abdominal incision. *Surg., Gynec. & Obst.*, 45: 678, 1927.
22. Spivack, J. L. Surgical Technic of Abdominal Operations. Chicago, 1936. S. B. Debour.
23. Starr, A., and Nason, L. H. Postoperative rupture of abdominal wounds. *J. A. M. A.*, 100: 310, 1933.
24. White, C. S. Innervation of muscles of the abdomen with reference to surgical incisions. *South. M. J.*, 28: 349, 1935.
25. White, W. C. Disruption of abdominal wounds. *Ann. Surg.*, 99: 34, 1934.

APPENDICITIS COMPLICATED BY GENERAL PERITONITIS

A METHOD OF TREATMENT THAT LOWERS THE MORTALITY RATE

E. G. WILLIAMSON, M.D., F.A.C.S., F.R.C.S. (ED.)

Instructor in Surgery, University of Pennsylvania Graduate School of Medicine; Associate Surgeon, Presbyterian Hospital

AND

L. M. RANKIN, M.D., F.A.C.S.

Chief of Service, General Surgery, Delaware County Hospital

PHILADELPHIA, PENNSYLVANIA

A REVIEW of the literature on acute appendicitis forces upon one the fact that the mortality rate is still much too high. Furthermore, we find that the neglected cases furnish most of the high rate—those that have gone on to perforation with general peritonitis. In this group of cases the mortality rate is variously given between 22 and 63.6 per cent. It is with this group alone we wish to deal, with the feeling that this high mortality is unnecessary.

It is our desire to report a series of thirty-two consecutive cases in this category, treated without a death. We do not include local abscesses or any other form of peritonitis where the mortality is naturally lower. Including these, our series of patients treated in the manner we are to describe is much larger (with the same result), but we wish to deal only with the more severe cases. We do not claim originality; as the literature is reviewed one wonders why the methods have not been more widely adopted.

It is generally conceded that in general peritonitis the problems presented are: (1) removing the focus of infection; (2) procuring as rapid an evacuation of the septic material from the abdominal cavity as possible; (3) preventing sepsis in the abdominal wound, which is supposed by some to be the vital factor in many deaths; and (4) preventing postoperative ileus.

As to the first, there seems to be little doubt at the present time that immediate removal of the appendix is desirable, after a reasonably brief period of preoperative preparation.

The second brings up the problem of drainage. The effect of posture on drainage in general peritonitis has occupied the mind of the surgeon since John G. Clark[1] presented his observations in 1897. He demonstrated:

1. That fluids and solids may pass through the lymph spaces of the diaphragm. He found the diaphragmatic portion of the peritoneum capable of absorbing much gross material.

2. That, as found by autopsy and upon the living patient, the drainage tract, no matter what material was used, always contained organisms of some kind.

3. That there was frequently fluid pent up in the drainage cavity which was sometimes forced into the general cavity.

4. That in many cases in which drainage was used, a sinus persisted six months or more.

He condemned external peritoneal drainage in most cases, and advocated postural drainage into the lymphatic system by elevating the foot of the bed 20 degrees, thus throwing the fluid against the diaphragm where it may be absorbed by the large open lymphatics. The disadvantage of this position soon became manifest and it was early abandoned.

Three years later, Fowler of Brooklyn reported results of nine cases of diffuse peritonitis treated in half-sitting posture.

This position, however, has a number of disadvantages. First, it is necessary to raise the patient so that the long axis of the abdominal cavity is at an angle of 60 or 70 degrees to the horizontal, before the spaces in front of the kidneys will drain into the pelvis. Again, this position tends to throw a decided strain upon a patient with an already weakened heart and lowered blood pressure. Furthermore, it favors gastric distention with its associated ill results. In this position the pelvis is lower than the pubic bones and the escape of pus takes place by siphonage.

Yates,[2] in 1905, reported some interesting observations on drainage. He experimented with all forms of drainage placed in different positions in the abdomen and was able to determine definitely; first, that all forms of drains were closed so that absolutely no drainage from the peritoneal cavity would take place after six hours; second, he concluded that drainage produces a flow of serum lasting from a few hours to two days, which is out of all proportion to the fluid in the cavity to be drained, but in exact proportion to the amount of drainage material inserted, showing that the serum was poured out as a result of the irritation of the drains; and third, he found that gauze will not drain pus or blood. Acting on these he condemned drainage of the peritoneum, as a rule, on the grounds, first, that it is impossible; second, it is depleting; and third, that pus and blood are not drained.

Coffey,[3] in 1906, published his "Principles and Mechanism of Abdominal Drainage," in which he stated that two mechanical principles are involved, gravity and capillarity. There are two steps in the process of drainage: first, bringing the fluid in contact with the drain; and second, delivery of the fluid to the surface. Gravity performs the first step in all intraperitoneal drainage, and, when postural methods are applicable, may also perform the second.

He suggested the well known Coffey dam drain, the principle of which is that two or more parallel tubes bound together work better than a single tube, which may become blocked. He showed that gauze will drain blood and pus because the excess liquid Yates mentioned liquefies and washes out the blood and pus; that the outer end of the drain must be as wide as the inner end and must also be lower than the depth of the cavity to be drained or in contact with similar material which is lower in order that capillary attraction will be continued. He also stated that drains must be left undisturbed for five to fourteen days until the area is completely walled off.

Among his deductions are the following: (1) A tubular drain drains perfectly a walled cavity, or will drain the peritoneal cavity under the influence of gravity if it does not become choked, but will not drain uphill, except when the fluid is confined. (2) There are three cavities or basins of the peritoneum to be drained; the right and the left flanks, separated from each other by the spinal column, and the pelvis, separated from the flanks by the psoas muscles. Either flank holds more fluid, and is an inch deeper than the pelvis, and its bottom is more than 4 inches below the top of the divide made by the psoas muscle, on which the appendix rests. The body must be elevated to an angle of 51 degrees to bring the bottom of the flank on a level with the divide and 60 or 70 degrees to drain properly by the Fowler position. The entire abdominal cavity can be drained by a lateral-ventral position. Coffey also showed that, "a line drawn from the center of the surface of the perineum to the tip of the shoulder will pass through the deepest part of the pelvis and flank." He called these lines the "right and left drainage lines." There are no independent cavities or basins between these lines, and the deepest points of the flanks are best reached externally to them.

In his conclusions Coffey stated: (1) Gravity is the most important principle in peritoneal drainage; therefore, drainage must reach the most dependent point of a cavity to be drained. The patient must be placed in the position that would naturally

cause the fluids in the peritoneal cavity to gravitate to the drain, always bearing in mind the anatomy of the three anatomic cavities or basins above described. (2) Gauze or capillary drainage is the most widely applicable and useful of all drains, if used in sufficient quantity to preclude its being choked by débris, and provided the drain is as large in circumference at its exit as it is at any point within the cavity, and provided also that it is in contact with an abundance of dressings on the outside. (3) If a surgeon remembers that his drain ceases to be effective in a few hours, he places it with the idea of removing septic fluid in the shortest time possible, according to the principles of drainage, and usually gets results. (4) If he is deluded by the belief that his drainage will continue to work for days, and that fluid will run uphill to get to the drains, he will usually place his drains accordingly, and consequently will be disappointed in drainage. (5) If a surgeon habitually removes gauze drainage before it is loosened by the natural process, a large percentage of his patients will have secondary sepsis, postoperative obstruction or hernia. (6) Drainage, except for the small precautionary cigarette or tubular drain, can rarely be safely removed until after the fifth or sixth day.

It is interesting to note that in 1905, Harbin[4] reported a patient treated in ventral decubitus as an aid to drainage for diffuse purulent peritonitis. He reported a patient, aged 44, drained with three iodoform gauze drains, who was put in the lateroprone position for thirty minutes, then shifted to his back for a short period, etc. "During the first six hours the drainage was very large in amount, saturating the dressings and sheets. The amount of discharge in this case by far exceeded what I have ever seen from any other abdominal wound and the amount of toxic material in the peritoneal cavity thus eliminated explained the rapid improvement of his symptoms and final recovery."

W. E. Leighton,[5] in 1914, reported a series of nine cases treated by using the prone position or ventral decubitus. Eight recovered, one died.

Roland Hill,[6] in 1916, reported fifty-seven cases of peritonitis or abscess formation treated in lateral or prone position with two deaths; forty-seven in Fowler position with five deaths—again including abscesses. He stated, "It is generally recognized that the posture in which patients are placed after operations for septic abdominal conditions is an important factor in their recovery. Unless sufficient drainage takes place in the first few hours the patient is likely to die from peritonitis or sepsis. Thus it will be seen that in the gravest surgical cases, it is of the utmost importance to secure at once an unobstructed outflow of all toxic fluids from the abdomen."

Glascock and Glascock,[7] in 1936, reported a series of eighty cases treated in ventral position with a mortality of 3.8 per cent.

Thus we see that the ventral position gives us the most rapid elimination of all the septic material from the abdominal cavity and fulfills requirements more nearly perfect than any other position. This rapid elimination of irritating material also tends to prevent postoperative adhesions and related postoperative complications.

As to the third requisite, prevention of sepsis in the abdominal wound, E. H. Poole,[8] in discussing a paper by Shipley and Bailey on the treatment of appendicitis complicated by peritonitis, in May, 1932, said, "In badly infected cases only sufficient sutures are introduced to close the peritoneum besides the drains; the rest of the wound is left open, for if it is closed above and below the drain, in a few days there is the typical colon bacillus infection, sloughing of the aponeurosis, etc. When the wound is left open there is no sloughing or necrosis, the wound rapidly granulates, no time is lost in convalescence and the incidence of incisional hernia is definitely diminished."

Gamble[9] advised the open treatment of peritonitis secondary to appendicitis, say-

ing, "This plan is founded on the assumption primarily that in 98 per cent of the cases of peritonitis, anaerobic organisms are present; that with the removal of the appendix the peritoneum is able to overcome the infection; that the infection of the abdominal wall plays a more important rôle in the causation of fatal sequelae than any other factor; that the closure layer by layer of the abdominal wall provides both the culture media and an excellent incubator for the growth of anaerobic organisms; that air is a specific for this type of infection; and lastly, that it is contrary to all surgical principles to close a fresh wound which has just been freely bathed in pus." He stated that Heyd in 1911 reported finding anaerobic organisms in 100 of 102 cases studied, while Jennings isolated anaerobic bacteria in 98 per cent of cases of acute appendicitis.

Anaerobic bacteria cannot grow in air. If this factor were taken into consideration in the treatment of general peritonitis complicating appendicitis, it is doubtful if there would be need for bacterins, serobacterins, vaccines, etc., against anaerobic bacteria.

Thus we see that the open wound is the answer to our third problem, the prevention of sepsis in abdominal wounds.

Paralytic ileus is treated mainly by prevention. Our ventral position aids considerably. In a series of major operations in the Presbyterian Hospital we have demonstrated the efficacy of prostigmin for this purpose. This drug maintains the muscular tone of the intestines by preventing parasympathetic paralysis. In large doses it is a parasympathetic stimulant. In our peritonitis cases we found no contraindications to its use and in two patients four months pregnant we used full doses with no effect on the gravid uterus. In our series of cases with the use of prostigmin both pre- and postoperatively, the patient on his abdomen, postoperative ileus was never a problem.

In reviewing and reporting these thirty-two cases from the Presbyterian Hospital (Philadelphia) and the Delaware County

Hospital (Drexel Hill), we find several points of interest. The youngest patient was 3½ years of age and the oldest 66 years. There were seven cases between 40 and 50 years of age, more than in any other ten year period. Between 10 and 20 years there were six cases; 20 to 30, five cases; 0 to 10, 50 to 60, and 60 to 70, had four each; there were two cases between 30 and 40 years.

There were eighteen males and fourteen females in the group. Thirty-one of the patients were white and one was black. This is in keeping with other reports which show the relative infrequency in the colored race.

The average onset of symptoms was three to four days before admission to the hospital. The lowest leucocyte count was 5,400, the highest 33,200, and the average 16,800. Eighteen patients gave a history of taking one or more laxatives before admission, citrate of magnesia, magnesium sulfate and castor oil were the most popular. On admission five gave a definite history of diabetes with average blood sugars of 230 mg. One patient had a blood sugar of 278. In addition the urinalysis in nine other cases was positive for sugar which later disappeared. All the patients were operated upon immediately, with either the median or lateral right rectus incision. The appendix was removed in every case. All were drained with one to three Penrose rubber-covered cigarette drains which were removed on the second or third day.

The pathologic report showed either acute gangrenous ruptured appendix or acute suppurative perforated appendix. The pus from the abdomen was cultured in twenty-four cases, with thirteen cases positive for B. coli, six positive for non-hemolytic streptococcus, two positive for Staphylococcus albus, and one positive for pneumococcus.

The peritoneum was closed down to the drains, the fascia and skin left open, and the wound packed with gauze saturated with tincture of merthiolate, azochlor-

CHART I

Case and Hospital	Age	Sex	Color	Days in Hospital	Onset before Operation	Laxative	W.B.C.	Urinalysis Sugar	Highest Postoperative temperature	No. Days before Normal Temp.	Pathologic Report	Culture	No. Drains	Condition of Wound at Discharge
S. D.C.	49	F	W	19	3	Mag. sulf.	15,000	Pos.	101	3	Ac. sup. perf.	....	1	Small area not healed
D. D.C.	60	F	W	23	2	Castor oil	12,200	Pos.	103.2	6	Ac. sup. perf.	Pneumococcus	2	Small area not healed
S. D.C.	49	F	W	35	2	Citrate	12,900	....	101	2	....	Nonhem. strep.	2	Small area not healed
Mc. D.C.	28	M	W	20	4	......	33,200	....	101	5	....	H. strep.	2	Small area not healed
Z. D.C.	65	M	W	22	3	Mag. sulf.	5,400	Pos.	102	6	Ac. gang.	....	2	Small area not healed
S. D.C.	54	F	W	43	3	Mag. sulf.	23,000	Pos.	104	12	Ac. gang.	B. coli	1	Healed
C. D.C.	10	F	W	27	7	Sal. hepat.	16,500	....	101	6	....	B. coli	1	Slight drainage
S. D.C.	66	M	W	20	7	Milk mag.	15,100	....	101.3	4	Ac. gang. perf.	H. strep.	1	Slight drainage
E. D.C.	14	F	W	12	3	Castor oil	13,600	....	101	5	Ac. gang. perf.	B. coli	1	Slight drainage
S. D.C.	31	M	W	13	3	Citrate	14,300	Pos.	101	4	....	B. coli	1	Slight drainage
R. D.C.	13	F	W	11	3	Citrate	15,900	....	102	3	Ac. sup. perf.	B. coli	1	Healed
R. D.C.	22	M	W	11	2	3 lax.	19,400	....	102.2	3	Ac. gang. perf.	....	1	Slight drainage
S. P.	24	M	W	39	2	One	19,450	....	101	5	Ac. gang. perf.	....	1	Small unhealed area
D. P.	6	F	W	14	3	One	17,750	....	102	8	Ac. gang. perf.	B. coli	2	Small unhealed area
P. P.	17	M	W	21	3	......	15,000	....	102.4	14	Ac. gang. perf.	....	1	Small unhealed area
C. P.	9	M	W	20	2	......	23,000	Pos.	101	3	Ac. sup. perf.	B. coli	2	Healed
W. P.	55	M	W	28	5	......	10,900	....	102.2	3	Ac. sup. perf.	B. coli	1	Small unhealed area
I. P.	12	M	W	14	2	......	23,000	....	102	5	Ac. gang. perf.	B. coli	1	Small unhealed area
K. P.	65	M	W	15	8	3 lax.	17,400	Pos.	103.3	1	Ac. gang. perf.	Nonhem. strep.	2	Healed
M. P.	41	M	W	19	2	......	14,800	Pos.	101	5	Ac. sup. perf.	Nonhem. strep.	2	Healed
M. P.	62	F	W	14	2	Citrate	17,900	Pos.	101	4	Ac. gang. perf.	....	2	Healed
P. P.	20	F	W	18	7	Cascara	16,650	Pos.	100.4	5	Ac. gang. perf.	....	2	Healed
Y. P.	58	F	W	32	7	......	19,000	Pos.	104	6	Ac. gang. perf.	B. coli	2	Healed
H. P.	46	M	W	12	2	Castor oil	22,700	....	102.3	7	Ac. gang. perf.	....	1	Healed
H. P.	44	M	W	15	2	......	11,500	Pos.	101.2	4	Ac. gang. perf.	B. coli	2	Healed
V. P.	17	M	W	16	2	......	19,300	....	101	6	Ac. sup. perf.	B. coli	1	Healed
C. P.	25	F	W	11	3	Citrate	13,350	....	104	5	Ac. sup. perf.	Staph. albus	2	Healed
K. P.	18	F	W	33	7	......	8,300	....	103	6	Ac. gang. perf.	Nonhem. strep.	2	Healed
K. P.	3½	M	W	17	7	......	20,150	....	103	4	Ac. gang. perf.	Nonhem. strep.	2	Healed
W. P.	46	M	W	29	2	......	16,100	Pos.	103.2	10	Ac. gang. perf.	Nonhem. strep.	2	Healed
V. P.	42	M	W	28	2	......	16,600	Pos.	103.3	5	Ac. gang. perf.	Staph. albus	2	Healed
B. P.	29	M	B	19	3	Mag. sulf.	19,080	....	102.3	6	Ac. gang. perf.	B. coli	3	Healed

amide, or similar antiseptic. Deep through and through retention sutures were put in but left loose in order gradually to pull the edges of the wound together as the infection disappeared. When the patient was returned from the operating room he was immediately put on his abdomen and the head of the bed elevated 8 to 12 inches. Routine treatment consisted in hypodermoclysis of saline and glucose and saline intravenously. An ampule of prostigmin was given every four hours and morphia was used for pain. Contrary to expectations the patients complained of very little discomfort because of the position.

It was very interesting to observe, as others who have used the ventral position have observed, that in each case the dressings rapidly became saturated with the drainage from the abdomen, necessitating frequent changing during the first twenty-four hours. The amount of discharge by far exceeded the amount in patients in the Fowler or other positions. This, because of rapid elimination of toxic material, undoubtedly accounted for the rapid drop in the temperature and pulse rate.

As soon as they were tolerated, water and then liquids were given by mouth; after thirty-six to forty-eight hours the patient was put on his back.

The highest postoperative temperature was 104.2 and the average time from the day of operation to the day the temperature reached normal was 5.3 days.

All were discharged from the hospital well after an average stay of 20.9 days and

the wounds were healed, with the exception of a small area at the site of the drains in a few of the patients. Twenty-one patients have been followed from one month to two years with excellent results. There was no sign of incisional hernia in any case and all the patients stated that they felt fine and had completely recovered from their operation.

CONCLUSIONS

Excellent results have been obtained with a negative mortality rate in general peritonitis complicating appendicitis by;

1. Immediate removal of the appendix, after a reasonably brief period of preoperative preparation.

2. Ventral position for the rapid emptying of septic material from the peritoneal cavity.

3. Open wound to prevent septic abdominal wall.

4. Use of prostigmin to prevent paralytic ileus.

REFERENCES

1. CLARK, J. G. J. Obst. & Dis. Women & Child., April-May, 1897.
2. GATES, J. L. Surg., Gynec. & Obst., Dec., 1905.
3. COFFEY. Principles and mechanics of abdominal drainage. Tr. West. Surg. A., p. 125, 1906.
4. HARBIN, R. M. M. Rec., p. 67, 1905.
5. LEIGHTON, W. E. J. Missouri State M. A., Dec., 1914.
6. HILL, R., Tr. West. Surg. A., p. 219, 1916.
7. GLASCOCK, H., and GLASCOCK, H., JR., Am. J. Surg., 34: 69, 1936.
8. SHIPLEY, A. M. and BAILEY, A. H. Ann. Surg., 96: 537–550, 1932.
9. GAMBLE, H. A. South. M. J., 29: 834, 1936.

TREATMENT AND MANAGEMENT OF BURN CASES*

H. JERRY LAVENDER, M.D.

Attending Dermatologist and Syphilologist, Cincinnati General and Jewish Hospitals

CINCINNATI, OHIO

MUCH has been written during the past fifteen years regarding the treatment of extensive burns, and especially of the use of agents such as tannic acid, gentian violet, acriviolet-brilliant green, silver nitrate, etc., alone or in various combinations. These chemicals, when applied to the burned areas, mix with the exudate to form crusts or leathery protective coverings. Ten to twelve hours, often fifteen to twenty hours, are required to obtain a leathery coating of the desired consistency. This form of treatment has been hailed by most investigators and generally accepted by the medical profession as a life-saving measure and the best treatment thus far advanced.

Advocates of this method claim in the main that it is a big factor in reducing shock, relieving pain, preventing loss of tissue and body fluids, helping to minimize the toxic stage, controlling sepsis, and speeding recovery. Almost without exception, however, the authors emphasize that the patient should be given the anti-shock measures commonly employed; that fluids be given in large quantities parenterally and by mouth (when possible); that narcotics or potent analgesics be administered to control pain; that provisions be made to control secondary pyogenic invaders; and that blood transfusions are very important. It would appear then, that the crust-forming method affords the patient a protective or leathery coat in the burned areas, but that the fluid loss, pain, toxemia and sepsis have to be met with other measures to enhance restitution and recovery.

No discrimination has been made up to this point between burns caused by fire and those caused by scalds. Although I do not consider the crust-forming method as the one of choice in either event, I might accede to its use in scald cases, but not in those caused by fire. It may be stated in general that most burns produced by scalds are not so deep as those caused by fire; also, that the extent and depth of an injury produced by scalds can be determined within a few hours after the insult, whereas several days, sometimes a week or longer is necessary before the same visualization can be accomplished after fire.

The reason for this is quite obvious. Unless the individual is subjected to a medium · of constant thermal potency (several minutes or longer), the damage or involvement after scalds will be much more superficial, due to the fleeting or disseminating property of the scalding liquid and its rapid loss of heat. Using the same time element, fire will produce a much more intense and a deeper burn. Also, it may be stated that, as a rule, scalds will manifest more inflammatory and bullous (exudative) reaction than fire burns, the latter cooking the flesh to such a degree that the vast majority of the areas are dry and remain so for several days. The exudative processes are seared off, so that there is very little loss of tissue or body fluids. The seared tissue thus acts in the same manner as a leathery covering, differing, however, in depth and consistency. What is the advantage gained by covering this already dry area with an additional coating?

It must be admitted that depth plays a rôle in the restoration of the tissues; also, that intensely cooked or burned tissue is, in the majority of cases, dead tissue. Dead tissue acts as a foreign body and restoration cannot take place without a separation and sloughing of this dead or gangrenous material. This is part of the body's defense

* From the department of Dermatology, Service of Dr. Elmore B. Tauber, Cincinnati General Hospital.

mechanism, and the leathery coat or crust does not, and for obvious reasons cannot, prevent this separation or sloughing. On the contrary, in the vast majority of cases, not only does the sloughing take place, but the crust, unless removed, slows the healing process and traps any pyogenic exudate underneath its tough covering.

If we accept the theory, of the origin of the toxic stage wholly or in part as a result of the absorption of split proteins, or if we wish to recognize the theory of the absorption of toxic material from secondary pyogenic invaders, we certainly could not prevent absorption of toxins from the deeply burned areas by manufacturing a crust over the burn. No matter how aseptic the technique in applying the crust, when the trauma of the burn involves tissues deeper than the epidermis, sloughing must ensue for proper healing and before complete restoration, with or without grafts, can take place. This refers, of course, to deep or "third degree" burns. Certainly no one would consider grafting an area which was obviously unclean. By the same token then, why resort to some measure in deeply burned areas which not only does not enhance the healing, but is contrary to all the laws of natural body defense?

It is also illogical to maintain that the leathery coat or crust makes the patient more comfortable and helps to tide over the interval until sloughing takes place. As stated previously, it usually requires several hours for a desirable coagulated area or firm crust to form. If the burn is extensive, with several areas involved, it means constantly changing the position of the patient in order properly to apply the crusting agent—certainly not a comfortable procedure for the patient. Even after the crust is firm and hard, it is much more difficult to change position from time to time for relaxation or further comfort. The castlike coating precludes this where extremities, and especially joint areas, are involved. If the crust has to be arranged in band-like formation, especially on the

extremities, it impedes circulation, acting as a tourniquet.

I fully realize that dehydration plays a big rôle through the rapid and enormous loss of blood and body fluids, but even in untreated extensive (area considered) superficial burns with a marked inflammatory and exudative process, the transudate or oozing of fluids into the burned areas will subside within a few hours. In fact, many of the cases are first seen several hours after the trauma is effected, and after most of the fluid loss has taken place. This loss must be met by parenteral administration of fluids. The leathery crust offers no help where the damage has already occurred or where the process of fluid loss has already greatly dissipated itself.

Therefore, the general supportive measures which are necessary in every case, i.e., intravenous solutions, blood transfusions, proper nourishment, etc., are the actual elements which build up the resistance, help the natural defenses and carry the patient through—not the coagulum.

Many of the statements in the foregoing discussion may seem dogmatic, but I have been forced to these conclusions after careful study and evaluation of many methods and after the supervision of over 2,000 burn cases.

My method of choice, in the vast majority of the cases, is the use of a modified, almost forgotten, water-bath or tubbing procedure.

The patient should be hospitalized. Freshly burned areas, that is, those seen within the first few hours, are not disturbed for at least ten to twelve hours. The patient is merely wrapped in a sterile sheet. This permits a more complete manifestation of the intensity of the burn, and the nature and extent of the injury are better visualized. In the interim, anti-shock measures are enforced. This includes the usual procedures of elevating the foot of the bed, warm covers and heat applications, warm intravenous solutions (the first usually containing 5 to 10 per cent glucose), stimulating drugs, absolute quiet, etc. The

patient is typed and matched as soon as possible and a blood transfusion given during the first twenty-four hours. Thereafter, blood is given often, and the pushing of fluids continued, depending upon the general condition. Suprarenal cortex extract has proven to be efficacious as a prolonged stimulant, and in many cases it was felt that it acted as a life-saving measure during both the shock and toxic stages.

No morphine or allied preparations are used before the patient voids. To begin with the shock produces an oliguria or even an anuria, and to tie up the excretions or prolong the anuria is unwise. If the patient is in a stage of excitement when first seen, or is quite restless or hysterical, one of the barbiturates will usually have the desired effect. Morphine is not necessary in early cases for the control of pain, since little or no pain is experienced after the first several minutes of the injury. The shock stage precludes this, and the deeper the shock the less pain, discomfort or restlessness. Only after the initial rest period and when the patient is completely out of shock, are the burned areas attended to.

After the administration of a mild narcotic, the patient is lifted into a large bathtub filled with hot water (100°F.), to which has been added about one pint of aqueous green-soap solution.* This is a much better preparation than the tinctured solution, in that it is non-irritating and, in fact, very soothing. There is only a minimal amount of discomfort, lasting just a minute or two when the patient is first tubbed. All the torso and extremities are completely submerged; a head rest with an inflated rubber ring will keep the head free. Canvas hammocks fastened with hook arrangements to the sides of the tub and adjusted with ease, may be used; they cause little or no discomfort due to the buoyancy of the body in the full tub.

* The soap solution is prepared in the same manner as the ordinary tincture of green-soap (U.S.P.) except that distilled water replaces the alcohol and the oil of lavender is eliminated. The preparation is, of course, filtered.

The individual is observed very closely and is permitted to remain in the soap solution for five to ten minutes only, depending upon the general condition and tolerance. He is then lifted to a sterile sheet, and a mechanical debridement of all blisters and loose tissue is done as aseptically as possible, using small pieces of sterile gauze. Care must be exercised to get the areas as clean as possible, especially the edges of the wounds. The whole cleaning up procedure should not consume over four or five minutes, even in an extensively burned patient. Dressings of warm dilute Burow's solution (2 ounces to the quart of water) are then applied to all areas involved. For this purpose sterile strips of old linen or cotton material should be used, and several layers applied. Gauze dressings are avoided to prevent firmer adherence or sticking to the wounds.

The dressings are kept wet continuously with the warm solution, but are not changed or removed until the next tubbing period, the following morning, at which time the patient is placed, dressings and all, in the bath containing the soap solution. After several minutes of soaking, the dressings gradually float away from the wounds without any lifting or pulling, and with no discomfort to the patient. The length of stay in the tub is now increased to twenty or thirty minutes, again depending on the tolerance, after which fresh dressings of dilute Burow's solution are applied in the same fashion as previously described without cleaning off the soap solution. The normal skin areas are hurriedly dried, either with a soft towel or a warm-air electric blower. This is a daily procedure.

Upon being returned to bed, the patient is observed for secondary shock, which, if it does occur, is usually very mild. After this the head and chest of the patient should be elevated to prevent hypostatic involvement, his position being changed every few hours for a similar reason. It is not unusual for the patient to fall asleep after the bath, the tubbing procedure act-

ing as a soporific; it is not uncommon for dozing to occur in the tub.

On the third or fourth day the patient will usually tolerate a full hour of tubbing and this may be increased gradually to several hours per session. The patient is then tubbed twice daily, in the morning and again in the late afternoon or evening. All but the head is completely submerged in order to prevent chilling, and the water should be kept at a relatively constant temperature. While he is in the bath, tap water may and should be given freely, and fruit juices may be added.

It is interesting to note with what pleasure patients anticipate the bath procedure; they are, quite often, reluctant to be removed from the tub. The water affords complete relaxation, relieving muscle spasm, pain or discomfort. Movements of the extremities can be accomplished with very little effort. Many patients prefer motion in order to release or remove the dressings themselves, and they become interested in this procedure.

With these daily mild exercises, contractures are, for the most part, either prevented or reduced to a minimum. It is rarely necessary to resort to some orthopedic appliance, but when necessary, this can be applied much earlier by the tub method because the nature and extent of the impending contracture or deformity can be anticipated sooner. Due to the fact that the dressings are not molested, and that they release themselves in the bath, the new granulation tissue is not disturbed: this is an important factor.

Another factor of even greater importance, is that the wounds are cleansed by the weak soap solution, which reduces secondary infection to a minimum. Occasionally a pyocyaneus infection is encountered and is treated with a 1 per cent solution of acetic acid. The odor of pyogenic involvement, so characteristic of burned areas, is absent. With cleaner areas, granulations form much faster, and the manifestation of epithelial islands may be watched from day to day.

In third degree burns, the gangrenous tissue separates and sloughs much more rapidly, and as this process takes place the base of the sloughing area remains clean. This makes for a much faster restoration and proliferation of epithelial tissue. Even after the sloughing is completed, the raw surface will in many instances develop small indented areas here and there, which turn out to be new islands of epithelium. As long as this process continues the tubbing is kept up until the areas are completely healed. If the healing process appears to be at a standstill, the patient is prepared for skin-grafting.

If grafting is necessary, the areas are almost ready for it without further preparation. Many of these cases have undergone grafting without dakinization, and with good results. If dakinization is necessary, it is only for one or two days, using weak solutions. If the area to be grafted manifests exuberant or hypertrophic tissue, the Dakin's solution will reduce them to skin level in most instances, providing that it is applied early and before hardening or complete organization of the scar tissue takes place.

If one area heals in advance of another, the wet dressings are replaced by 5 per cent boric unguent in the healed areas. Vaseline-gauze may also be used. Most investigators will agree that in facial burns, even intensive ones, restoration and healing will take place much more rapidly and with less scarring than in other areas. The wet dressings with Burow's solution will suffice to bring about a proper response in most cases. Occasionally a 2 or 3 per cent ammoniated mercury ointment may be used to prevent or restrain secondary pyogenic infection.

Infants and children respond unusually well to the bath method of treatment. Their handling is, of course, relatively simple in comparison with adults, as they may be lifted in and out of the tubs with ease. Adults, however, very soon become adapted to the manipulation, and their anticipation of the soothing and relaxing

effects of the bath more than compensates for the effort involved in getting them there. Water toys help greatly, in amusing children and in getting them to move various members of the body as they play.

CONCLUSIONS

Accurate comparative statistics for different methods of treatment are not available; comparisons may be made only when like conditions arise. The location, the extent of cutaneous involvement, and the depth of the burn, the age of the patient and the burn itself, together with the general management and treatment, must be considered, as all play an important rôle. One patient with a relatively superficial and limited involvement, may die of a complication such as pneumonia, erysipelas, or post-surgical scarlet. Another, extensively and intensively involved and with a poor prognosis, may sail on to a relatively uneventful recovery. Therefore, deductions as to efficacy of therapy can be made only on a general basis, after thorough practical application and experience.

I feel that the bath or tubbing procedure, as outlined, far surpasses any other form of therapy advocated. Its advantages over the protective-crust or coagulum method are manifold, and may be summarized as follows:

1. Epithelial proliferation and new granulations not disturbed, thereby enhancing recovery.

2. Burned areas are kept much cleaner.

3. No coagulum to trap pyogenic infection.

4. Separation and sloughing of dead tissue occurs more rapidly, leaving cleaner base.

5. Contractures and adhesions are prevented by movement of involved parts; orthopedic intervention may be started earlier.

6. Areas are ready for grafting earlier.

7. The procedure is soothing, comfortable, and practical.

8. No irritation or deleterious effects, attributable to the soap solution, have been observed in any case.

THE TREATMENT OF BURNS*

H. D. COGSWELL, M.D.   AND   CLAYTON SHIRLEY, M.D., F.A.C.S.

TUCSON, ARIZONA   TYLER, TEXAS

EXTENSIVE burns constitute one of the most serious and complicated conditions to be cared for in the realm of traumatic surgery. The course of a burn can be prolonged and disheartening to both the patient and the physician. Burn contractures, especially those involving the head, face, or neck, may result in deformities so severe as to cause an unfortunate victim to lose all semblance of a human being. With these cases there is not only physical damage but enough mental anguish so that an entire personality may be changed. People who have become hideously scarred from burns become self-conscious, invertive, and often develop a definite psychoneurosis. The treatment, therefore, should be thorough in order to prevent deformities and to repair, by plastic surgery, those deformities which do occur.

The largest amount of constructive research and treatment in this field has been accomplished in the past few decades. Before this the treatment of burns was largely empirical.

Children are more frequently burned than adults. Of the fatal burns occurring annually in the United States, 45 per cent are in children under 6 years old.[1] At the Children's Hospital in Detroit the greatest number fell in the age group of 1 to 3 years.[2] Children and the aged tolerate extensive burns poorly. Low[3] reported that scalds caused 57 per cent and matches or fire 20 per cent of the burns of the children admitted into the Children's Hospital at Boston. The prevention of burns should be stressed, particularly in localities where open heating systems (gas and gasoline heaters) are commonly used. A publicity campaign stressing the danger of burns and

means of avoiding them would be a valuable form of prophylactic treatment.

Methods of treatment of burns are legion, and it is most probable that they began with the discovery of fire by man. Burns must have been one of the first conditions ever to receive treatment. Until 1925 when Davidson[4] advanced his theory of toxemia and advocated tannic acid treatment there had been little reduction in the high mortality rate of burned individuals. Tannic acid was not new, however, as the Chinese were using infusions of tea leaves for burns as early as the sixth century B. C., tannic acid from the leaves accounting for the efficacy of this treatment. In 1890, an anonymous author published a paper in the Pittsburgh Medical Review recommending the use of 5 per cent tannic acid on burns.[2] Unfortunately, this plan of therapy was not adopted, and until 1925, pastes, oils, ointments, paraffin, etc., constituted the accepted treatment. Willems and Kuhn,[6] reported that 1,206 burned individuals had been treated by 386 physicians and given forty-seven different types of treatment. The majority of methods used in treating burns have been aimed at allaying pain and to a large extent this still holds true.

Burns have been variously classified, Dupuytren naming as many as eight degrees. A simplified classification, now commonly used, groups major and minor burns. A minor burn is one involving not over 15 per cent of the body surface and so situated that the patient may remain ambulatory. A burn of the face involving so large an area should be classified as a major burn due to its location and the constant care necessary. Major burns are those involving over 15 per cent of the

body surface. Both of these groups are divided into three degrees. The first degree burn shows simple erythema of the burned surface but no vesication or necrosis. A second degree burn produces destruction of the epidermis with erythema, vesicle, and bulla formation. A third degree burn varies from complete destruction of the skin to involvement of the deeper structures.

Another type of burn which should receive consideration may, for simplicity's sake, be termed the "inhalation burn." This is caused by inhaling hot fumes or caustic chemicals, and is an insidious and dangerous form of injury, often fatal, whose seriousness is frequently not recognized until late. The scope of this paper does not include a discussion of this latter type of burn but confines itself to the treatment of burns of the body surface.

Davidson's tannic acid treatment was based on the theory that there was absorption of a proteid substance from the burned area, causing a toxemia. This view was supported by the experimental work of Robertson and Boyd,[7] but Underhill and his co-workers[8,9] found that there was a greatly diminished rate of absorption from the burned area. It was also found that there was an increase in capillary permeability, causing a shift of the water balance and resulting in anhydremia due to loss of fluid from the blood to the injured area. In experimental animals a third degree burn of one sixth of the body surface caused a fluid loss of 70 per cent of the total blood volume in twenty-four hours. During this period there was a decrease in the blood cholorides, a depletion of adrenalin in the blood, and a distinct increase in hemoglobin. Blood urea and non-protein nitrogen remained normal and there was no acidosis.

The cause of shock and the associated symptoms during the first forty-eight hours following a burn can be explained by the anhydremia which produces an increase in blood viscosity. The result is a diminished heart output, a slowing of the circulation and anoxemia, with incomplete excretion of the toxic waste products of metabolism. The want of oxygen may be partially responsible for the degenerative changes of the liver, adrenals, spleen, and kidneys observed after fatal burns.

About the second or third day following a burn, symptoms develop which cannot be ascribed to the loss of body fluids. At this stage definite signs of toxemia appear, namely, malaise, chills, fever which may range as high as 105 to 106 degrees (of the "picket-fence" type), drowsiness, restlessness, muscle twitching, and periods of irrationality. Adequate fluids, although essential in this period, do not relieve these symptoms and findings. It was reasoned by Firor and Aldrich[5] that an extensive infection of the burned surfaces could be responsible for the toxemia. By culturing the burned areas they found that there was very little bacterial growth in the first twelve hours. Following this relatively sterile period, staphylococcus, B. coli, and streptococcus were found in 100 per cent of the burned cases, with the last named predominating. As the signs of toxemia increased a greater concentration of streptococci was found.

An understanding of the physiology of burns clarifies the treatment necessary. The treatment of minor burns should consist of: (1) alleviation of pain; (2) prevention and treatment of infection; (3) orthopedic care (as in burns of the hands, antecubital or axillary areas) to prevent contraction deformities; (4) reconstructive surgery when indicated. In major burns the treatment is roughly divided into the following stages: (1) treatment of shock, pain, and anhydremia; (2) prevention and treatment of infection; (3) prevention of exhaustion; (4) orthopedic care to prevent flexion deformities; (5) reconstructive surgery.

MINOR BURNS

The most important phases in the treatment of minor burns are the prevention of infection and the relief of pain. Early it is often difficult to determine the depth of the

burn, as erythema and pain may be the only evidence of thermal injury. Later, vesicles may appear or the epidermis may slough, showing the presence of a third degree burn. Due to the fact that the diagnosis of a second or third degree burn is obscure when a burn is first seen, oils, greases, and ointments should not be applied as a remedy. These greasy agents, aside from their analgesic effect, have no proved therapeutic value. Grave reactions have occurred following the use of some of these due to the patient's sensitivity to the drug.[10]

If it can be definitely ascertained that the burn is of first degree, a sterile vaseline gauze dressing may be sufficient. Mercurochrome 5 per cent,[11] gentian violet or ferric chloride 5 per cent[12] covering the erythematous area are in the line of good treatment. Later if blebs appear the field has been kept sterile and no greasy substances are present to invite infection. The blebs should be aspirated by a sterile needle introduced into their base. A compression bandage is then applied to prevent the protective epithelium from being loosened. Often the epidermis of the blister will become revascularized with a complete and rapid cure.[13] A second or third degree burn of a part such as the hand or foot should be treated with tannic acid and silver nitrate (described below). This simplifies dressings, is a worthy analgesic, and prevents infection. If pain is severe a suitable dose of morphine or codeine should be given immediately. A burn situated over a joint may be painful due to motion, and immobilization of the joint is indicated in these cases. The treatment of infection, prevention of contractures, and application of reconstructive surgery is similar to that described under major burns.

MAJOR BURNS

Most major burns occur among the indigent. This is due in large part to housing conditions, heating units, and the fact that these families tend to have more children. These individuals can least afford

hospitalization and nursing care, yet they need careful treatment and laboratory facilities which can be obtained only in a hospital. Often by the time these patients arrive in a hospital they are thoroughly chilled from transportation in wet clothing, or they are covered with a greasy substance that insures infection.

Soon following a major burn the patient usually presents a definite clinical picture characterized by intense pain, lowering of the blood pressure, a fast pulse of poor volume, rapid shallow respirations, anxious face and restlessness. These signs are due to primary shock and probably to vasodilatation of the splanchnic area.

There are some patients with severe major burns who do not show primary shock, are fairly comfortable and appear to be in good condition when first observed. After an interval of approximately six to ten hours the symptoms described above appear. This condition is known as secondary shock and is due to depletion of the blood fluids. This form of shock frequently appears in patients who have rallied from primary shock and were seemingly improved.

Statistics show that 60 per cent of deaths from burns occur in the first twenty-four hours.[14] Treatment therefore should be instituted early. As soon as a patient with a major burn is seen, all wet clothing should be cut and removed. If an attempt is made to save clothing by pulling arms out of sleeves, pulling clothes over the head, etc., a portion of the epithelium may be torn away and a raw area liable to infection created. Morphine should be given, since pain, as well as fluid loss, is a prime factor in shock. The patient is placed in warm blankets to exclude the air. If a patient feels chilled or shows a subnormal temperature, external heat should be applied and the victim hurried to the nearest hospital. It is dangerous to treat the burn itself in any manner until the period of shock is well under control. Patients with major burns who do not appear to be in shock should be treated as if shock were present—it invari-

ably will occur sooner or later. Delayed shock is often more severe than initial shock and is more difficult to treat; consequently it should be averted. The shock accompanying burns is treated in the same manner as "surgical shock" by giving fluids, stimulants, morphine, external heat, and elevating the lower portion of the body.

As soon as the patient is safely out of shock the burned area should be treated. If there has been an application of grease this must be removed by placing the patient in a tub of warm water in a *warm* room and gently cleansing the area with green soap and water. Following this the patient is moved to sterile sheets and a light anesthetic is administered. Pentothal sodium has been used intravenously in a few cases when smaller areas have been involved, but a gas or gas-ether anesthetic is generally more satisfactory. When there is an associated "inhalation burn" inhalation anesthetics are contraindicated due to the edema of the lungs, and a rectal anesthetic such as avertin should be used. A complete debridement of the burned surface can be accomplished by using a soft scrub brush and scrubbing all burned areas with green soap and water. It will be found that the burned epithelium can be rapidly removed by this method. The entire injured surface is then copiously irrigated with sterile saline or water. Any remaining grease can be removed with ether.

Some writers have advocated irrigating the injured areas with mild antiseptics such as hexyresorcinal, merthiolate solution, etc. We have used these preparations, but it is an expensive procedure, and although no harm is done by their application it seems wise to await more confirmation of their value before advocating their employment.

Following the irrigation the patient is dried with sterile towels, and 5 per cent tannic acid is sprayed over the burned areas with an atomizer. A solution of 10 per cent silver nitrate is applied over this. A fresh cotton swab should be used for each application to prevent tannic acid being carried into the silver nitrate container. The rapidity of the formation of a coagulum is surprising. Bettman's[14] tannic acid-silver nitrate treatment is vastly superior to the use of tannic acid alone. When tannic acid is used alone, it must be sprayed on every fifteen minutes for a period of twelve to twenty-four hours before a proper eschar is formed. By using tannic acid and silver nitrate in combination, this can be accomplished in fifteen minutes. The almost instantaneous sealing of the burned areas inhibits pain, stops the fluid loss, protects the raw area from infection, diminishes the incidence of delayed shock, simplifies the nursing problems, and leaves a pliable coat over the joints. Tannic acid alone has other disadvantages; when infection sets in under the thick eschar there is no local evidence of what is going on. It is also painful, often cracking, when applied over a joint. It has been shown that epithelium can be destroyed by tannic acid,[16] and a red inflammatory area exists in the surrounding zone of a burn following its use. When the tannic acid-silver nitrate solution is used this is not found, nor is the albuminuria present that is commonly associated with burns. There is apparently no danger from argyria as no evidence of silver absorption has been noted.[15]

After completion of the tan the patient is placed in a "heat cradle," consisting of an electric lighted tent which holds the sheets from the patient, assures the patient ample freedom, and keeps the bed comfortable and warm. A thermometer is suspended from the top of the tent in order that the temperature may be controlled. At from 85 to 88°F.

It is most important that the patient receive adequate fluids and chlorides. One liter of fluid should be given for every 25 pounds of body weight every twenty-four hours. Hemoglobin determinations are an invaluable aid in determining the amount of anhydremia present and how successfully it is being combatted by fluid administration. To maintain the water balance in children the continuous intravenous drip is

very practical. Anchoring a number 4 ureteral catheter in one of the ankle veins permits limited motion of the leg. Adults are given normal saline with 5 to 10 per cent glucose intravenously at intervals until the necessary amount can be taken orally. Nausea and vomiting occasionally occur during the first forty-eight hours, the latter sometimes being obstinate. If 500 c.c. of a 3 per cent saline solution is given, very slowly, intranveously, and these symptoms are completely abated. When the hemoglobin reading is nearing normal, care should be taken to prevent too large an amount of fluids being given. Children should be especially watched for edema. Edema may not only be due to an excessive amount of fluids but may be the result of a salt block due to retention of chlorides. If a salt block exists 1,000 c.c. of distilled water with 10 per cent glucose given intravenously will immediately relieve the edema and associated symptoms.

After several days the patient's temperature should recede and approximate normal. If this has not occurred and signs of toxemia are present, a search should be made for an infection. Burns which have had grease applied, burns around the perineum, burns involving both sides of the body so that the patient is forced to lie on a burned area, or burns whose crust has been loosened, are prone to infection. Fluctuation of the eschar, tenderness, and localized elevation of the tan usually reveal the location of purulent material. The tan should be removed in this region, and the septic area allowed to drain. Wet packs of ¼ per cent chlorazene solution are used to clean the wound of gross infection.

Until the burned areas are entirely healed supportive treatment is essential. Patients with extensive infected burns will eventually succumb unless given every aid in the surgeon's armamentarium. These patients show progressive anemia, anorexia, weakness, and gastrointestinal symptoms; unless they are carefully treated, they waste away and die from exhaustion or from some concurrent disease. The most valuable medicines that we have to combat this decline are blood transfusions and an adequate diet. Frequent small blood transfusions are an excellent tonic, much more beneficial than the large transfusions given as a last resort. "Last resort" transfusions in burns are seldom life saving. We favor the use of citrate blood transfusions as the patient's veins are saved. In addition, it is more convenient, avoids the necessity of moving the patient, can be better controlled, and has all the advantages of whole blood in these cases. Transfusions are indicated when there is a fall of hemoglobin or other evidence of anemia, when the food intake has been limited, when the healing process is slow or at a standstill, or when the plasma protein is near 5.0 Gm. per cent.

The diet should be high in calories, easily assimilated, and of a high vitamin and mineral content. During the healing period the caloric intake of adults should exceed 2,500 calories daily. A sick individual cannot be forced to eat a routine diet and should be allowed to choose his menu as long as it contains the necessary elements. Sixty per cent of the body sulfur is contained in the skin and the sulfur derived from a diet containing eggs definitely promotes epithelization.

It is feasible to remove the coagulum as soon as the epithelium has regenerated. This usually begins taking place at the end of seven to ten days, at which time the tannic acid-silver nitrate coat will be seen to curl at the edges. No definite time can be set for removing the eschar as this factor depends on the individual healing factor and the depth of the burn. Bits can be removed from time to time, but unless there is an infection present or the eschar is loosened by underlying moisture the crust should remain at least three weeks if healing has not taken place.

When any unhealed or infected areas remain, the area should be skin grafted as soon as possible. The ideal site for a skin graft is a bed of vascular granulation tissue free from gross infection. In most instances the constant use of ¼ per cent chlorazene

packs will promote healthy granulations. In certain individuals a purulent fibrous exudate appears over the burned surface. In these cases hot water soaks and manual removal of the exudate improve the granulations. Even though handled properly, some burns reach an indolent stage when there is little or no proliferation of epithelium or granulation tissue. Ultra-violet therapy is a benefit in these cases.[17] If this is not available the heat of an ordinary light bulb will increase circulation. Transfusions are an adjunct in stimulating tissue growth, diminishing infection, and should always be given when the healing process is slowed. Scarlet red ointment, cod liver oil ointments, or applications of oxyquinoline sulfate scarlet R gauze[14] promote epithelization and may be used in this indolent stage. They should not, however, be applied over infected granulation tissue.

Some burns are not seen by a surgeon until long after it has been treated by home remedies, ranging from soot to advertised salves and greases. By this time the patient is debilitated and the burn infected, covered with a purulent crust, and looking like an old ulcer surrounded by an area of cellulitis. Blood transfusions, proper diet, removal of the crusts by warm water soaks and the use of chlorazene packs aid the patient's condition and render the injured region amenable to skin grafting.

Areas of exuberant granulation tissue may form and prevent the ingrowth of epithelium. If such a condition exists the granulations can be controlled by cauterizing them with lunar caustic and applying pressure dressings of mesh gauze held in place by an ace bandage.

Second or third degree burns over joints invariably cause scar contractures or flexion deformities if not properly treated. Their prevention is less difficult than their cure and, for this reason, any joint in the region of the burn should be placed in extension and splinted.

The optimum time to begin reparative work is before scar tissue forms contractures. When the granulation tissue has a beefy red, clean appearance with a thin blue line of epithelium growing at the edges of the raw area, conditions are right for skin grafting. Since whole thickness grafts are disfiguring to the donor site and prone to fail because of poor viability, Ollier-Thiersch grafts which bisect the papillary layer of the skin seem most satisfactory. Pinch grafts have their value in burns with infected granulation tissue where only small clean areas are present to receive a graft, or where burns are so extensive that the donor sites are too small or poorly located for Thiersch grafts to be obtained. Although it has been shown that homoplastic grafts do not remain permanently,[18] homografts are at times indicated. In massive burns, when the patient's condition continues to decline, it is dangerous to increase the amount of raw body surface by removing skin for autografts. By covering the granulating surface with grafts from another individual of the same blood group the raw areas are temporarily healed. During this period the patient is saved the disastrous effects of septic absorption. Autoplastic grafts can be applied in two to five weeks when the homoplastic grafts disappear.[19] The technique of skin grafting and reconstructive surgery is not discussed in this paper as this is an entire subject in itself.

SUMMARY

1. The use of greases, ointments, or salves is contraindicated in the treatment of burns.

2. Due to the shift of the water balance in burned patients, large amounts of fluids are indicated to prevent delayed shock and symptoms of anhydremia.

3. The burned areas are not to be treated in any manner until the patient is well out of shock. Then they are treated like surgical wounds, by debridement, tannic acid 5 per cent, and silver nitrate 10 per cent applied to the injured surfaces with aseptic technique.

4. Exhaustion and debilitation must be avoided by frequent blood transfusions, proper diet, and care of the infected area.

5. Skin grafting should be done as soon as the granulations permit and flexion deformities prevented by extension splints over joints.

REFERENCES

1. Pack, G. T. Thermic injuries. Cyclopedia of Medicine, Philadelphia, 1934. F. A. Davis Co.
2. Penberthy, G. C. Treatment of burns. *New England J. Med.*, 214: 306, 1936.
3. Low, M. B. Tannic acid-silver nitrate treatment of burns. *New England J. Med.*, 216: 553, 1937.
4. Davidson, E. C. Tannic acid in treatment of burns. *Surg., Gynec. & Obst.*, 41: 202, 1925.
5. Aldrich, R. H. Preoperative and Postoperative Treatment. Philadelphia, 1937. W. B. Saunders Co.
6. Willems, J. D., and Kuhn, L. P. Burns. *Am. J. Surg.*, 34: 254, 1936.
7. Robertson, B., and Boyd, G. L. Toxemia of severe superficial burns. *J. Lab. & Clin. Med.*, 9: 1, 1923.
8. Underhill, F. P., and Kapsinow, R. Alleged toxins of burned skin. *J. Lab. & Clin. Med.*, 16: 823, 1931.
9. Underhill, F. P. Significance of anhydremia in extensive superficial burns. *J. A. M. A.*, 95: 852, 1930.
10. Queries and Minor Notes. *J. A. M. A.*, 108: 992, 1937.
11. Turner, A. C. Contribution to treatment of burns. *Brit. M. J.*, 2: 995, 1935.
12. Coan, G. L. Ferric chloride coagulation in treatment in burns. *Surg., Gynec. & Obst.*, 61: 687, 1935.
13. Bancroft, F. W. In Christopher's Textbook of Surgery. Philadelphia, 1937. W. B. Saunders Co.
14. Bettman, A. G. Tannic acid-silver nitrate treatment of burns. *Northwest. Med.*, 34: 46, 1935.
15. Bettman, A. G. Rationale of tannic acid-silver nitrate treatment of burns. *J. A. M. A.*, 108: 1490, 1937.
16. Taylor, F. Misuse of tannic acid. *J. A. M. A.*, 106: 1144, 1936.
17. Gatch, W. D., and Trusler, H. M. Use of ultraviolet light on the preparation of infected granulation tissue for skin grafting. *Surg., Gynec. & Obst.*, 50: 478, 1930.
18. Trusler, H. M., and Cogswell, H. D. Question of homoplastic skin grafting. *J. A. M. A.*, 104: 2076, 1935.
19. Bettman, A. G. Homogenous Thiersch grafting as a life saving measure. *Am. J. Surg.*, 39: 156, 1938.

TRAUMATIC TORSION OF THE OVARIAN PEDICLE*

A MEDICOLEGAL STUDY WITH PRESENTATION OF CASES AND REVIEW OF THE LITERATURE

HYMAN SNEIERSON, M.D., F.A.C.S.,

Attending Surgeon, Binghamton City
Hospital

BINGHAMTON, NEW YORK

JULIUS SCHLESINGER, M.D.

COLLEGE POINT, NEW YORK

AND

A. E. GOLD, ESQ.

Justice of the Supreme Court, State of New York

BINGHAMTON, NEW YORK

"TRAUMA, industrial and non-industrial is increasing so rapidly that it now occupies almost a dominant position in the practice of surgery," (Lazenby[96]).

With the ever widening scope of compensation insurance as well as the tendency of attributing nearly every disease to accidental injury, traumatic medicine and surgery has increasing importance to every member of the profession. Many conditions have only recently been accepted as being sometimes caused by injuries. Among these are ulcers of the stomach,[70-78] appendicitis,[1-19] certain heart conditions,[69] and even cancer, although this is still being contested. It has become the tendency of patients, often inspired by a desire for financial reward, to claim that illness resulting almost any time after the injury was initiated at that time. The final decision is often made by a jury of laymen and precedent established without scientific basis. It is consequently apparent that there is need for further study and reporting of every case which is caused or claimed to be caused by injury.

Personal experience in two cases of torsion of the ovary in which trauma was accepted by insurance carriers as the initiating factor has stimulated the following paper.

All case reports of tumors of the ovary, including those of torsion from 1925 to 1936 at the Binghamton City Hospital, were examined. The literature of twists of the ovary was also carefully studied. In view of the fact that our interest lay almost entirely in the traumatic angle, a review of the literature aside from that relevant to the subject has been omitted. The reader is referred for more complete data to the excellent articles by Wong[67] and Haines and Edgerly.[37]

It also seemed to the writers that the rôle of trauma as an etiologic factor from the legal point of view would contribute to the value of this study. We have, therefore, incorporated a brief but fairly complete statement as to what in general constitutes legal liability in any pathology following an alleged trauma and also a discussion of our cases from the legal viewpoint.

Torsion of the ovary has been reported since 1842[37] and is a fairly common occurrence in surgical practice. The etiology, however, has never been scientifically investigated. This is probably due to the fact that the posture which is present in man and which apparently plays such a part in producing the condition is not present in the animals available for experimentation. Study in the main has been along clinical lines with rationalization following the finding of the torsion.

Haines and Edgerly[37] mention as etiologic factors sudden movements such as falling or slipping, exertion such as cough-

ing and sneezing, alternate distention and evacuation of the bladder and even the passage of feces through the rectum. Pressure of a pregnant uterus or emptying of the uterus may also be exciting causes. Unequal growth in a tumor[23] is mentioned in slow torsion.

In the early editions of Graves' "Textbook of Gynecology," trauma was one of the causes mentioned. The latest edition, however, has omitted this. Trauma is also mentioned in many other articles examined by the writers. Wong[67] lists trauma as a cause, but classifies under this heading such things as jolts, falls, sudden changes of posture, and so forth, which are not so classified by other writers. Only four cases in the literature available to the writers were found in which a single direct and definite trauma was the precipitating factor in the ordinary sense of the word. Darner[32] cites the case of a child who was kicked in the abdomen. This was followed by pain and a progressive condition found at operation to be due to torsion of an ovary. Wong cites one case in which the condition followed a fall. He also cites a case under the heading of trauma which followed the lifting of heavy luggage. Cohen[27] reports a case in which sharp pain followed the lifting of a mattress. At operation torsion of a fimbrial cyst was found.

Two factors must be present in every case of torsion of the ovary; first, a free tumor and second, an elongated pedicle.[23, 37] Even then torsion occurs in only a few cases. Graves[36] states that twisting occurs in 10 to 20 per cent of ovarian tumors. Payr (quoted by Haines and Edgerly[37]) showed by a model that increase in the venous pressure of the pedicle without increase in the arterial pressure tends to produce torsion without other contributing factors. Anything, therefore, which causes a slowing up or blocking of the venous return may cause torsion. "Torsion of the pedicle of an ovarian cyst may begin suddenly and in the space of a few hours cause acute symptoms. It is probable, however, that in the majority of the cases the torsion tends to occur slowly, little by little, extending over a period of weeks or months or years; then even a very slight increase in this slow torsion may suddenly bring on acute symptoms."[37]

The rapidity with which symptoms occur following the torsion is of paramount importance in determining the etiology. The first sign which calls the attention of the patient to trouble in the acute cases is pain. We have carefully tried to ascertain from previous reports and books on pathology just when this first occurs. Due to complete absence of experimental evidence the subject is a matter of conjecture. The only conclusion possible is that interference with circulation if severe enough and of sufficient rapidity will cause pain. That the onset of pain does not necessarily mark the initiation of vascular pathology was brought out in the analogous condition of mesenteric thrombosis by Warren and Eberhard. The pain is caused only by circulatory interference and this may occur anytime during the process. This fact is important in any determination as to etiology. In examining case reports cited there is usually a history of repeated attacks of pain occurring over a period varying from hours to years. In some cases the beginning of the attack was definite and the condition progressive. In nearly all, however, careful questioning elicited the history of similar but subsiding attacks.

Without repeating the reports in detail the following conclusions may be drawn from the literature on the subject. Pain indicates fairly rapid impairment of circulation. The pain may be transient, the impairment being sufficiently corrected to relieve this symptom, either by untwisting completely, or to some degree. The twisting may persist and be compensated, as indicated by recurring attacks of pain coupled with increasing size of the tumor and abdomen. The number of times that twisting and untwisting may occur and the exact factors which cause them remain conjectural. If the interference with circulation is sufficient the condition is pro-

gressive and irremediable. Surgery is always indicated at this time.

At this point we submit a typical case report by von Graff.[64] This embodies the factors mentioned above and also involves the question of trauma as an etiologic factor. Two days following an auto accident, a patient near term was delivered of a dead child. Soon after delivery she experienced pain in the right lower quadrant. This pain persisted off and on with exacerbations at the menstrual periods and with increasing size of the abdomen. At operation one year later, a large twisted ovarian cyst was found. The cause given by the writer for the torsion was the delivery. No history was given of any injury to her abdomen at the time of the accident and trauma was not considered as a possible

There were five deaths in this series. These all occurred in complicated cases. One was a case of ruptured ovarian cyst with marked intraperitoneal hemorrhage simulating an ectopic pregnancy. The wound broke open within twenty-four hours and was immediately resutured with a fatal result. Another death occurred in one of the seven cases of infected cysts. The other three were operated for intestinal obstruction. Two of these had had previous operations and the ovarian pathology was found at the laparotomy. The other case was diagnosed as twisted ovarian cyst with intestinal obstruction. At operation, adhesions between the bowel and the ovarian tumor were so dense that resection was necessary. End-to-end anastomosis by means of a Murphy button was done.

TABLE I
OVARIAN TUMORS 1925–1936
Binghamton City Hospital

Type of Ovarian Tumor	Number Unilateral	Number Bilateral	Deaths	Complications
Simple cyst	150	14	4	Torsion in 9 cases.
Pseudomucinous cyst	5	1	0	One case of pseudomyxoma peritonei.
Multilocular cyst	8	0	1	Torsion in 3 cases.
Dermoid cyst	7	2	0	
Cystic carcinoma	6	3	0	
Serous papillaryous cystadenoma	7	3	0	Torsion in 2 cases.
Fibrosis	3	0	0	Torsion in 1 case.
Fibroma	6	0	0	
Chocolate cyst	4	1	0	
Sarcoma	1	0	0	
	197	24	5	

cause. If claimed, trauma might reasonably have been accepted as a causative factor.

During the ten year period (1925–1936) 221 cases were operated on. The greater number were simple cysts and the types and percentages generally fell within the ranges reported elsewhere.[23,66] There were two cases of ruptured cysts; two were gangrenous; pregnancy was associated with eight of the simple cysts; seven ovarian cysts were infected; there was one case of pseudomyxoma peritonei.

Included in the 221 cases were fifteen cases of twisted ovarian cysts. There was no mortality in this group.

In analyzing the fifteen cases of torsion of ovarian pedicle, no great variation from previously reported series was noted.[23,66] The greater proportion was in married women (nine) and on the right side (twelve). The age range and the pathology followed the literature.

The types of tumors found were at variance with the larger series reported by

TABLE II
TORSION OF OVARIAN CYSTS 1925–1936
Binghamton City Hospital

	Case Number	Married or Single	Age	Presence of Trauma	Onset Sudden	Onset Gradual	Duration	Association with Pregnancy	No. of Prev. Preg.	Presence of Mass	Preoperative Diagnosis	Operative Findings
1.	A5214	M	16	..	x	..	2 wks.	..	..	x	Twisted right cyst.	Torsion of simple right ovarian cyst 1½ times.
2.	95203	M	45	..	..	x	3 yrs.	..	4	x	Twisted right cyst.	Torsion of simple right ovarian cyst with hemorrhage.
3.	89300	S	28	..	..	x	4 mo.	..	..	x	Twisted left cyst.	Torsion of normal left ovarian cyst with hemorrhage.
4.	97192	M	36	..	x	..	6 mo.	..	..	x	Twisted right cyst, intestinal obstruction.	Torsion of simple right ovarian cyst with adherent bowel and intestinal obstruction.
5.	70000	M	23	..	..	x	6 mo.	..	..	x	Strangulated right ovarian cyst.	Torsion of multilocular right ovarian cyst.
6.	61191	M	29	..	..	x	3 yrs.	..	..	..	Undetermined.	Torsion of simple right ovarian cyst with gangrene.
7.	89354	M	42	x	..	x	7 wks.	..	4	x	Twisted right cyst.	Torsion of simple right ovarian cyst with hemorrhage.
8.	A511	S	43	..	..	x	5–6	..	..	x	Twisted right cyst.	Torsion of simple right ovarian cyst with gangrene.
9.	94822	S	31	x	x	..	2 da.	..	..	..	Twisted right cyst.	Torsion of multilocular right ovarian cyst.
10.	36090	S	27	..	x	..	5 da.	..	..	x	Twisted left cyst.	Torsion of simple left ovarian cyst with hemorrhage.
11.	39433	M	37	..	..	..	......	..	..	..	Twisted right cyst.	Torsion of papillary cystadenoma of right ovary.
12.	47693	S	16	..	x	..	6 da.	..	..	..	Perforated appendix.	Torsion of simple right ovarian cyst with hemorrhage.
13.	54030	M	31	..	..	x	4–6 mo.	x	..	x	Pregnancy and cancer of omentum or pedunculated fibroids.	Torsion of multilocular ovarian cyst.
14.	46219	M	38	..	x	..	......	x	..	..	Right ectopic.	Torsion of papillary cystadenoma of right ovary.
15.	45842	S	13	..	..	x	......	..	..	..	Appendicitis.	Torsion of simple right ovarian cyst.

Bernstein,[23] inasmuch as nine were simple cysts, three papillary cystadenomata and only one was a solid tumor. The solid tumor was 12 by 9 by 6 cm. and consisted entirely of a moderately hard encapsulated mass which was hemorrhagic. It had a solid beefy appearance and grossly was infiltrated with blood. Microscopic examination showed a normal ovarian stroma infiltrated with large quantities of blood. Infarction was present in some places. Baron[21] also reports torsion of a normal ovary.

The symptoms varied from two days to three years or more in duration. Where a careful history was elicited this period could be stretched indefinitely. This is of great importance in attempting to state when the trouble started and in formulating an opinion as to the immediate cause. In thirteen cases the onset of symptoms was without apparent immediate cause. Two patients gave a history of pain following trauma. Both of these were accepted legally as such and compensation was awarded.

The first case was in a middle aged obese woman who was riding in the back of an automobile with a dishpan containing produce in her lap. The front wheel of the car was struck by a train, throwing her violently forward and forcing the side of the dishpan into her abdomen. Following this she experienced severe pain in her abdomen with vomiting and was put to bed for four days. She improved for the time being and was able to do her work off and on for seven weeks, with occasional periods of bed rest. During this period she was repeatedly examined by the surgeon for the railroad company who considered the condition due entirely to bruising of the abdominal wall. At times she had intense abdominal pain with vomiting but these were of short duration. At no time did she have any bowel or urinary disturbance. The day before admission the pain became more severe and she vomited constantly. Her bowels moved, however, without difficulty. She became rapidly worse and was therefore hospitalized. On admission examination revealed a large abdominal mass which was diagnosed as a twisted ovarian cyst. At operation, a torsion of an ovarian cyst the size of a large grapefruit was found. The circulation was impaired to such an extent that the mass was almost black. On section, the ovary was found to be filled with dark blood.

In this case, a violent direct external blow was given to the abdomen. This was followed immediately by symptoms suggesting internal injury. These symptoms persisted and finally culminated in an acute abdomen necessitating surgical intervention. The pathology at operation was clearly consistent with the trauma sustained. We feel that the injury first produced either a hemorrhage into the cyst or an edema of the tumor which caused impairment or retardation of the venous return. This venous obstruction caused a gradual progressive further impairment of the circulation with the twisting caused by such pathology, as demonstrated by Payr (quoted by Haines and Edgerly[37]). In our opinion the blow did not cause a twisting of the ovary immediately, such as would occur in striking a ball on a string. The twisting came secondarily. The initial pain was probably due to bruising of the abdominal wall as well as to injury to the ovarian cyst. Later, the pain of torsion was superimposed. We do not feel that the torsion was complete at first. The terminal pathology could not have lasted seven weeks without gangrene and peritonitis. There was, however, sufficient trauma to cause the production of the pathologic condition necessary to initiate this torsion. The history, aside from the trauma, could be reproduced from other cases in our series.

The second patient presented an entirely different picture. An unmarried waitress of 31 years, while lifting a tray, experienced a sudden sharp pain in her right side radiating to the right thigh. The pain was well localized, and persisted off and on for two days. She was in bed during this period. Before admission she developed frequency and tenesmus. Her menstrual history was essentially negative. The bowels moved without difficulty. A diagnosis of acute appendicitis was made and the patient referred to the Binghamton City Hospital. On admission she had a normal leucocyte count and no elevation of temperature. Examination showed muscle spasm in both lower quadrants. Rectally a mass the size of a small orange was noted in the cul-de-sac. A diagnosis of twisted ovarian cyst was made and substantiated at operation. The cyst was

twisted two and one-half times. The tube was also twisted but without interference to its circulation. The color of the ovary was good. Impairment of circulation was apparently restricted to venous compression.

This patient had been a waitress for many years. The lifting of the tray was a frequent daily occurrence with her. There was no unusual circumstance associated with the pain, such as slipping or tipping of the tray, necessitating sudden change in posture.

The pain was, of course, due to impairment of circulation caused by twisting of the pedicle. Just why the pain should have occurred at the time stated is the point in question.

Gardner (in Dean Lewis' "Practice of Surgery" states that torsion *is said to follow some unusual exertion*, such as reaching up high. In this way the recti muscles are put on an unusual stretch, thereby obtaining a new grip on the cyst and rotating it. In our case the cyst was apparently not of sufficient size, to enable this phenomenon to happen. Again referring to the analogy of a ball on a string, it seems impossible that the cyst was twisted two and one-half times by one act.

Two inferences are possible. The first is that the pain was caused by immediate twisting with instant impairment of circulation. We cannot in the absence of experimental evidence disprove this hypothesis. The absence of marked interference of circulation two days later, even with the pedicle twisted two and one-half times, would suggest that the condition had not changed materially since its onset.

The second assumption is that the torsion was already present before sufficient pain was experienced to attract the patient's attention to it. If that is true, any one of the factors enumerated as causing torsion of the cyst, such as straining, stretching, turning over in bed or any motion, might change the position of the cyst sufficiently to cause symptoms. According to this hypothesis, very little change in position would cause the occurrence of subjective complaints. In fact, once the condition had progressed to a

certain point, it was irreversible and symptoms must ensue without regard to the acts or position of the host at the time they appeared. In other words, the pain was probably coincidental rather than the result of the alleged trauma. Aggravation of the existing pathology is the most that can be scientifically accepted.

MEDICAL DISCUSSION

Trauma causing torsion of the ovary must be divided into two types, direct and indirect. In the direct type, the injury is applied over or about the area involved. The exciting factor in the indirect type therefore includes all other types of trauma which might possibly cause torsion of the pedicle.

Direct violence has been accepted as a cause in many abdominal conditions, notably in peptic ulcer[70–78] and appendicitis.[1–19]

Where the diagnosis is accepted in these conditions, writers, both here and abroad, have insisted upon the following conditions:

1. The patient must have been in good health just prior to the accident.

2. No previous accident of a similar nature must have occurred.

3. There must be a definite history of trauma followed by pain, nausea and vomiting, which is progressive to the time of operation.

In cases of peptic ulcer and appendicitis, the trauma is understood to have caused an abrasion in the mucous membrane or an injury to the tissues which is demonstrable either grossly or microscopically. In cases of torsion of the ovary, overt injury to the ovary or its pedicle is not generally understood to be necessary, the trauma merely causing a twisting and not an abrasion or contusion.

In the seventh case listed in Table II there was in our opinion a contusion of the ovary with secondary torsion. We feel that in traumatic torsion of the pedicle caused by direct violence, injuries to the tissues grossly or microscopically evident are also a necessary concomitant. We believe that twisting of the pedicle in the manner that a

ball twists when struck on the side is not a satisfactory scientific medical explanation. We believe that the twisting occurs secondarily after interference with the circulation caused by the bruising and edema of the primary tissue according to the experiment of Payr.

Indirect violence is a vague term and scientifically is impossible to prove. Turning, twisting and indeed any act, normal as well as abnormal, may be so classified. The pathology found may be at a great distance from the site of the injury. The factors of predisposition, abnormal preëxisting pathology and a host of other questionable points are involved.

Indirect violence as a cause of peptic ulcer and appendicitis has been accepted by some writers, at least for medicolegal purposes. In these cases, however, injury to the tissues of the stomach and appendix is understood. Crohn and Gerendasy[72] state that peptic ulcer may be caused by violence in a distant part of the body. They explain this along the principles of contrecoup.

Shutkin and Wetzler[15] accept indirect trauma as a cause of appendicitis. They insist, however, that this be violent, acute, or unexpected.

Indirect trauma in cases of torsion of the ovary cannot be compared with peptic ulcer and appendicitis caused by indirect violence, since no definite injury to the tissues of the ovary or pedicle is understood.

The present writers, in surveying the subject, were struck by the similarity between torsion of the ovary[20–68] and torsion of the intestine or volvulus.[79–95] If we excluded all but cases involving the pelvic colon or sigmoid, we had an almost similar condition. A comparison of the etiology from the standpoint of trauma proved valuable. Both organs are in the pelvis and at the same anatomic level. In both, an unusual length of the mesentery (pedicle) and an abnormal mobility are considered essential for torsion. The etiologic factors are also similar. Tumors, mesenteric cysts, fecoliths, foreign bodies, pregnancy, constipation, direct violence and many other causes are given as the initiating factors in volvulus as well as in ovarian pedicle torsion.

In volvulus, as in torsion of the ovary, Sir Berkeley Moynihan[90] noted the tendency to recurrence. Richardson[92] stated that the rotation may be insufficient to cause intestinal obstruction, the condition causing recurring or chronic disability. Brenner stated that of twenty-eight cases, seventeen had previous symptoms caused by partial torsion. The torsion varied from 90 degrees to four complete turns. The analogy is therefore very close with the exception of intestinal obstruction, either partial or complete, in the case of volvulus.

Stoian, Tedorescu and Florescu[94] cited a case of acute volvulus following violent exertion. The patient had been in good health previously. There was, however, a loop of ileum strangulated in the anterior iliocecal fossa as well as the volvulus of the intestine.

Essau,[81] in 1875, von Bergman,[95] in 1900, and Garre,[84] in 1901, reported cases following severe muscular exertion.

Brenner[79] reported two cases of volvulus following direct trauma.

A comparison of volvulus and torsion of the ovary, however, is unfortunately of little assistance in finally determining the question of trauma as an etiologic factor. The same conditions and resulting torsion occur in both. The exact initiating factor, on the other hand, still remains unknown. Direct trauma as a cause must be accepted in both. In each, actual injury to the tissues is understood and the resulting pathology logically follows.

Indirect trauma in the case of torsion of the ovarian pedicle seems less logical than that of volvulus of the sigmoid. Peristalsis or movement is normally present in the sigmoid and an increased amount due to unusual or abnormal conditions can be understood to cause torsion. The ovarian pedicle on the contrary plays a passive role only. Twisting must be caused entirely by outside influences.

In direct traumatic torsion as in traumatic appendicitis, etc., the same postulates should hold: (1) previous good health; (2) no previous attack of a similar nature; and (3) a definite history of trauma followed by pain and progressive symptoms of torsion of the pedicle, leading to operation or death.

The conditions which will warrant the conclusion of torsion caused by indirect trauma cannot be definitely stated. From the literature and our own experience we feel that there is no such clinical entity as indirect violence initiating sufficient immediate torsion of the ovarian pedicle to give the pain syndrome demonstrated in the cases cited.

Indirect trauma as an aggravating factor in torsion which is .already present does correspond with known facts. Consequently twisting, lifting, slipping and so on must be accepted as causes of indirect trauma, when aggravation is alleged.

DISCUSSION OF LEGAL PRINCIPLES

The purpose of the law is to award just and reasonable compensation "for the natural and proximate consequences of the wrongful act."[97]

The principle is substantially the same in industrial accidents. "Injury and personal injury mean only accidental injuries arising out of and in the course of employment and such disease or infection as may naturally and unavoidably result therefrom."[98] There must be a causal connection between the accident and the ultimate condition for which compensation is claimed. The connection, when proved, establishes liability not only for the immediate consequences of the injury but also, for all other conditions flowing unavoidably therefrom. It has been held, for example, that there was a sufficient causal relation between an accidental injury and death when it appeared that the deceased was suffering from a dormant disease which became activated and brought on such mental derangement that by his conduct he contracted diphtheria followed by pneumonia.[99]

It is also fundamental that an accidental injury need not be the sole cause of the ultimate condition. It is sufficient if it is established to be an operative contributing cause.[100] It has been decided, for example, that the amputation of a leg following traumatic infection to the foot was compensable even though it appeared that the claimant had an existing diabetic condition without which the infection probably would have been checked.[101]

Legal liability is not limited to the effect of an accidental injury upon a person in sound health. A dormant or existing condition which is activated, accelerated, or aggravated by an accident is also compensable.[102] "The negligent actor may be liable for harm to another although a physical condition of the other which is neither known or should be known to the actor makes the injury greater than that which the actor as a reasonable man should have foreseen as a probable result of his conduct."[103] In a recent New York case, for example, compensation was awarded for restriction in abduction and rotation of a shoulder and atrophy of the left upper arm although it appeared that the claimant had a dormant arthritic condition which was aggravated by the accident.[104]

It is apparent that the applicable principles of law in this field must necessarily be broad and general. In the final analysis, each case will depend upon its own facts. There is generally no difficulty. The causal connection is usually clear when credible evidence is offered of the accident, its severity, previous integrity of the wounded part (except when the claim is activation or aggravation), the appearance of symptoms sufficiently close to the injury as to warrant the inference of cause and effect, and identity of the injured area with the ultimate condition for which compensation is claimed.

It must also appear that the condition is one which can conceivably be caused by trauma. The decisive factor is of course, medical evidence. The difficulty is that the etiology of many conditions is either uncer-

tain or unknown. Nevertheless, the law in dealing with the facts of everyday life expects medicine to furnish an opinion which will do justice with as much certainty as is possible in the light of medical knowledge at the time.

CASE I. In this case, an automobile in which the claimant was riding was struck by a train, causing direct injury to the abdomen with the appearance of symptoms immediately. The causal connection is clear. It is agreed that torsion of the ovary may be caused by direct trauma. It is not important from a legal standpoint whether the trauma caused the twisting immediately or secondarily. The decisive factor is that the torsion was the proximate consequence of the injury.

CASE II. In this case, the claimant apparently suffered a strain in her right side while lifting a tray. Liability was accepted by the insurance carrier, apparently because sudden strains have been held to be accidents within the scope of the Workman's Compensation Law.[117]

The difficult question is whether there was any causal relation between the strain and torsion of the ovary. The question is obviously medical and not legal.

It seems clear that the strain by itself did not cause the twisting. Apparently, torsion was already present. The effect of the strain may have been to aggravate the condition. If that is true, the case was compensable under the principles of law stated above.

SUMMARY

1. Traumatic torsion of the ovarian pedicle is a distinct entity.

2. The mechanism of the direct type is scientifically logical and when found, evidence of injury, either gross or microscopic is present.

3. Indirect trauma cannot be accepted as the initiating cause of the condition but may aggravate a torsion which is already present.

4. Legal liability may rest upon either type of trauma.

5. From a legal standpoint, it is immaterial whether the trauma is the sole cause of the torsion or merely aggravates or accelerates an existing condition.

REFERENCES

Traumatic Appendicitis

1. BEHAN, R. J. Traumatic appendicitis. *Ann. Surg.*, 85: 263–268 (Feb.) 1927.
2. BISSELL, A. H. Appendicitis, traumatic. *Arch. Surg.*, 17: 672–675 (Oct.) 1928.
3. Budapest Letter. Appendicitis and trauma. *J. A. M. A.*, 92: 999 (March 23) 1929.
4. EICKEN, F. Relation of trauma to appendicitis. *Zentralbl. f. Chir.*, 57: 3095, 1931– Abst. in *J. A. M. A.*, 96: 911 (March 14) 1931.
5. LUPTON, C. H. Traumatic appendicitis. *Virginia M. Monthly*, 52: 250–252 (July) 1925.
6. MAES, URBAN, and McFETRIDGE, E. M. Traumatic appendicitis. *Am. J. Surg.*, 30: 478–483 (Dec.) 1935.
7. McCARTHY, P. A., and McGRATH, J. L. Traumatized vermiform appendix. *Am. J. Surg.*, 39: 148 (Jan.) 1938.
8. Medicolegal. Appendicitis, trauma and workmen's compensation. *J. A. M. A.*, 97: 876 (Sept. 19) 1931.
9. Medicolegal. Traumatic appendicitis. *J. A. M. A.*, 99: 2290 (Dec. 31) 1931.
10. Medicolegal. Appendicitis attributed to a strain (Syde's case, Maine, 142 A. 777). *J. A. M. A.*, 92: 1705 (May 18) 1929.
11. MILSON, L. Traumatic appendicitis. *Wisconsin M. J.*, 31: 100–101 (Feb.) 1932.
12. MORTENSEN, H. N., and BRAY, L. Case of appendicitis with unusual features. *M. J. Australia*, 2: 346 (Sept. 15) 1928.
13. OPPENHEIMER, L. S. Traumatic appendicitis. *J. Florida M. A.*, 11: 193–194 (Jan.) 1925.
14. SABAWALA, B. P. Trauma and appendicitis. *Brit. M. J.*, 1: 630 (April 14) 1923.
15. SHUTKIN, M. W., and WETZLER, S. H. Traumatic appendicitis. *Am. J. Surg.*, 31: 514–520 (Mar.) 1936.
16. SMITH, T. H. Trauma and appendicitis. *Brit. M. J.*, 1: 17 (Jan. 6) 1923.
17. WHITE, W. D. Traumatic appendicitis. *Minnesota Med.*, 14: 733–736 (Aug.) 1931.
18. WILD, H. W. Trauma and appendicitis. *Brit. M. J.*, 1: 467 (March 17) 1923.
19. *Zentralbl. f. Chir.* Traumatic appendicitis. *J. A. M. A.*, 55: 217–226 (July 21) 1938.

Twisted Ovarian Cysts

20. ARNIM, L. C. Pathology and treatment of complications of ovarian cysts. *Texas J. Med.*, 30: 30–35 (May) 1934.
21. BARON, C. Torsion of the normal ovary. *J. A. M. A.*, 102: 1675 (May 19) 1934.
22. BEALL, F. C. Strangulation of sigmoid flexure by pedicle of an ovarian cyst. *Ann. Surg.*, 95: 854–855 (June) 1932.
23. BERNSTEIN, P. Torsion of the pedicle in ovarian tumors. *Arch. Surg.*, 25: 787–794 (Oct.) 1937.

24. BESSESEM, A. N., JR., and BESSESEM, D. H. Case of ovarian cyst with twisted pedicle. *M. J. & Rec.*, 128: 414 (Oct. 27) 1928.

25. BLOCK, F. B., and MICHAEL, M. A. Torsion of the normal fallopian tube. *Am. J. Obst & Gynec.*, 26: 268 (Aug.) 1933.

26. BRIDE, J. W. An investigation of a series of 100 ovarian tumors. *Edinburgh M. J.*, 32 (March) 1930.

27. COHEN, H. Report of two cases of fimbrial cysts; one with twisted pedicle and gangrene complicating pregnancy and one simulating an ectopic gestation. *Am. J. Obst. & Gynec.*, (Aug.) 1921.

28. COUNSELLER, V. S. Complications of dermoid cysts of the ovary; chemical hysterectomy. *S. Clin. North America*, 10: 151–154, 1930.

29. CURTIS, P. M. Cyst of ovary in child aged three years. *M. J. Australia*, 1: 497 (April 21) 1938.

30. DAVISON, C. L. Twisted ovarian cyst; a procedure to prevent fatality from embolism. *Am. J. Surg.*, 27: 79 (Jan.) 1935.

31. DELIGTISCH, E., and VERNICK, S. W. Ovarian dermoid cyst with twisted pedicle simulating renal colic; a case report. *M. Rec.*, 145: 205–206 (March 3) 1937.

32. DARNER, H. Torsion of the normal fallopian tube. *Am. J. Obst. & Gynec.*, 11: 368 (March) 1936.

33. DOWNER, I. G., and BRINES, O. A. Torsion of undiseased uterine adnexa in virgins. *Am. J. Obst. & Gynec.*, 21: 665 (May) 1931.

34. ELBIM, A., and MERIGOT, L. Torsion of ovarian cysts in children. *Rev. franç. de gynéc. et d'obst.*, 27: 358, 1932.

35. GOPALAN, T. C. An ovarian dermoid with twisted pedicle. *Indian M. Gaz.*, 59: 83–84 (Feb.) 1934.

36. GRAVES. Gynecology, 3rd and 4th Ed. Philadelphia, 1924. W. B. Saunders Co.

37. HAINES, C. E., and EDGERLY, W. S. Torsion of pedicle of ovarian cyst in young children. *M. Rec.*, 144: 121–125 (Aug. 5) 1936.

38. HELVESTINE, F. J. Torsion of the pedicle of ovarian cysts. *Am. J. Obst. & Gynec.*, 9: 259–262, 1925.

39. KASMAN, L. P. Simultaneous gangrene. Twisted ovarian cyst and acute gangrenous appendicitis. *Am. J. Obst. & Gynec.*, 28: 614–615 (Oct.) 1934.

40A. KESSLER, H. H. Traumatic appendicitis. *Am. J. Surg.*, 42: 555 (Dec.) 1938.

40B. KICKHAM, C. J. Ovarian cyst with twisted pedicle in girl nine years of age. *New England J. Med.*, 212: 665 (April 11) 1935.

41. KINCAID, H. L., and ANDREWS, E. Twisted ovarian cyst in five year old child. *Am. J. Obst. & Gynec.*, 15: 207–212, 1928.

42. KOUCKY, J. D. Torsion of ovarian cysts; diagnostic value of referred pain. *S. Clin. North America*, 8: 663–666, 1928.

43. KOUCKY, J. D. Ovarian dermoids; a study of 100 consecutive cases. *Ann. Surg.*, 81: 821–832, 1925.

44. LEONARDO, R. A. Torsion of hydrosalpinx. *New York J. Med.*, 36: 879 (June) 1936.

45. MACRAE, R. C. Twisted ovarian cyst in child. *Brit. M. J.*, 1: 906 (May 17) 1930.

46. McEACHERN, A. Torsion of the normal fallopian tube. *Brit. M. J.*, 3813: 190 (Feb. 3) 1934.

47. NICHOLSON, N. J. Twisted ovarian cyst with absence of uterus. *Lancet*, 1: 249 (Feb. 4) 1933.

48. OBADALEK, W. Twisted ovarian cysts in children. *Monatscbr. f. Kinderb.*, 48: 419–433 (Nov.) 1930.

49. PFEIFFER, D. B., and SMYTH, C. M. Ovarian cyst twisted on its pedicle with carcinoma of the sigmoid discovered incidentally. *S. Clin. North America*, 6: 207 (Feb.) 1926.

50. PRICHARD, J. A. Bilateral twisted ovarian cysts. *Brit. M. J.*, 1: 1077 (June 13) 1925.

51. PRIDE, C. B. Ovarian cyst on twisted pedicle. *West Virginia M. J.*, 25: 736–738 (Dec.) 1929.

52. RAINEY, W. R. Tumor of the ovary with twisted pedicle, occurring in a child two and half years old. *Ann. Surg.*, 79: 879–883.

53. REUBEN, M. S. Torsion of ovarian cysts in children. *Arch. Pediat.*, 43: 54–59 (Jan.) 1926.

54. ROBINS, C. R. Ovarian cyst with torsion of the pedicle; a case in woman 77 years old. *South. M. & S.*, 97: 549–551 (Oct.) 1935.

55. ROBINSON, J. S., and GROVE-WHITE, J. H. Torsion of large dermoid ovarian tumor. *Brit. M. J.*, 2: 700, 1925.

56. SCHEFFEY, L. C. Report of a case of spindle-celled sarcoma of the ovary with a twisted pedicle (removed by Dr. Brooks Anspach). *Am. J. Obst. & Gynec.*, 12: 896 (Dec.) 1926.

57. SCHWARTZ, L. S. Torsion of ovarian carcinoma. *Am. J. Obst. & Gynec.*, 9: 375–381, 1925.

58. SHELDON, D. Complete torsion of tube and ovary complicating pregnancy. *Am. J. Obst. & Gynec.*, 31: 682 (April) 1936.

59. SICHEL, M. S. Torsion of pedicle of dermoid cyst. *Minnesota Med.*, 9: 481 (Aug.) 1926.

60. STEEL, W. A. Torsion of ovarian cyst in children. *Brit. M. J.*, 21: 798 (Oct. 31) 1931.

61. STEVENS, I. H. K. Ovarian cyst with twisted pedicle in a child. *Brit. M. J.*, 2: 1083–1084 (Dec. 27) 1930.

62. THOREK, M. Case of ovarian fibroid with thrice-twisted pedicle. *Illinois M. J.*, Dec. 1924.

63. VANDERVEER, E. A. Strangulated ovarian cyst simulating appendicitis. *Tr. A. A. Obst. & Gynec.*, 29: 371–375, 1916.

64. VON GRAFF, E. Torsion of pedicle in ovarian cysts following delivery. *J. Iowa State M. S.*, 25: 118–124 (March) 1935.

65. WALKER, M. A., and RILEY, R. B. Abdominal pain due to torsion of hydrosalpinx. *J. Kansas M. S.*, 37: 462 (Nov.) 1936.

66. WIENER, S. A study of the complications of ovarian tumors. *Am. J. Obst.*, 72: 209, 1915.

67. WONG, A. I. H. Ovarian cysts with torsion of pedicle. *China M. J.*, 42: 625–644 (Sept.) 1928.

68. ZELLHEOFER, H. W. K. Gangrenous ovarian cyst with twisted pedicle; report of case. *Proc. Staff Meet. Mayo Clin.*, 11: 11 (Feb. 12) 1936.

Traumatic Heart Trouble

69. BECK, C. S. Address before Binghamton Academy of Medicine. December 15, 1936.

Traumatic Ulcer of Stomach and Duodenum

70. BOTTCHER, R. On subcutaneous injuries of the mucous membrane of the stomach. *Arch. f. Verdauungskrank*, 29: 40, 1922.

71. COBET, R., and STERN, R. Traumatism and ulcer perforation, discussed on the basis of an "expert opinion." *Med. Klin.*, 25: 1786–1788 (Nov. 15) 1929.

72. CROHN, B. B., and GERENDASY, J. Traumatic ulcer of the duodenum and stomach. *J. A. M. A.*, 100: 1653 (May 27) 1933.

73. EUSTERMAN, G. G., and MAYO, J. G. Traumatic peptic ulcer. *Am. J. Surg.*, 26: 74 (Oct.) 1934.

74. HAUSBRANDT, F. Traumatic ulcers of the stomach. *Wien. klin. Wchnschr.*, 1: 681–684, 1932.

75. RAYMOND, F., and CHENE, P. Chronic ulcer of the stomach of traumatic origin. *Press. méd.*, 40: 40–549 (April 9) 1932.

76. RUGE, E. Industrial accident and gastric ulcer. *Arch. f. klin. Chir.* 163: 728 (March 10) 1931.

77. SILBERSTEIN, A. On the genesis of traumatic gastric ulcer. *Med. Welt*, 1: 149–152 (March 5) 1927.

78. WIEGAND. Causative relation between accident and gastric injury, presumably gastric ulcer. *Ztschr. f. ärztl. Fortb.*, 24: 297–302, 1927.

Traumatic Volvulus

79. BRENNER, E. C. Total volvulus. *Am. J. Surg.*, 16: 34 (April) 1932.

80. DEAVER, and MOGOUR. Main contributing factor constipation or obstipation. *Surg., Gynec. & Obst.*, Jan. 1927

81. ESAU, V. Volvulus of the intestine. *Deutsche Arch. f. klin. Med.*, 16: 474, 1875.

82. FANUCCI, M. Intermittent volvulus of stomach. *Policlinico (Sez. chir.)*, 39: 35 (Jan.) 1932.

83. FURBER, T. M. Chronic volvulus of sigmoid. *M. J. Australia*, 851, 1930.

84. GARRE. Volvulus of the sigmoid flexure. *München. med. Wchnschr.*, 48: 520, 1901.

85. GRIDNEV, A. Volvulus of intestines. *Arch. f. klin. Chir.*, 174: 712, 1933.

86. HYMER, A. Recurrent attacks of volvulus of sigmoid. *Am. J. Surg.*, 4: 443 (April) 1928.

87. HABERER, H. Partial volvulus of stomach through diaphragm. *Deutsch. Ztsch. f. Chir.*, March 1926.

88. LIGAT, and OVEREND Recurrent volvulus of pelvic colon. *Brit. M. J.*, 2: 7 (July 1) 1933.

89. MATRY, M. Recurring volvulus. *Presse méd.*, vol. 6 (Jan.) 1931.

90. MOYNIHAN, B. Abdominal Operations, Vol. II. Philadelphia, 1926. W. B. Saunders Co.

91. RANKIN, BARGEN, BUIE. The Colon, Rectum and Anus. Philadelphia, 1932. W. B. Saunders Co.

92. RICHARDSON, E. P. Graham's Surgical Diagnosis, Vol. II. Philadelphia, 1930. Saunders.

93. ROWLANDS, R. P. Recurrent volvulus of pelvic colon. *Brit. M. J.*, 1: 289 (Feb. 16) 1929.

94. STOIAN, TEDORESCU and FLORESCU. *Rev. san mil.*, quoted in *J. de chir.*, April 1926.

95. VON BERGMAN, A. Diagnosis and treatment of intestinal occlusions. *Arch. f. klin. Chir.*, 61: 885, 1900.

Trauma

96. LAZENBY, A. D. *Surg., Gynec. & Obst.*, 64: 393 (Feb. 15) 1937.

Rules of Law

97. Wallingford v. Kaiser, 191 N. Y. 392, 397.

98. New York State Compensation Law, Sec. 2, Subd. 7.

99. Malgieri v. General Electric Co., 258 N. Y. 620.

100. Ehrgott v. N. Y., 96 N. Y. 264.

101. Snyder v. Union Mills, Inc., 32 St. Dept. (N. Y.) 380.

102. Mazzarisi v. Ward, 170 A. D. (N. Y.) 868.

103. Restatement of the Law of Torts, Sec. 461.
The comment under this section reads:
"The rule stated in this Section applies not only where the peculiar physical condition which makes the other's injuries greater than the actor expected is not known to him, but also where the actor could not have discovered it by the exercise of reasonable care, or, indeed even where it is unknown to the person suffering it or to anyone else until after the harm is sustained. A negligent actor takes the risk that his liability will be increased by reason of the actual physical condition of the other towards whom his act is negligent."

104. Kazen v. Steinberg Press Co. Inc., 250 A. D. (N. Y.) 797

105. Watkins v. New Brunswick Restaurant, 242 N. W. (Neb.) 439; McDonough v. Scott, 3 Cal. I.A.C. 225; Contra In Re Gardner, 4 Ohio Com. 21.

106. Pilloud v. 350 Park Avenue Co., 16 Ind. Bull. (N. Y.) 225.

107. Grant v. Narlion, 1 Cal. I.A.C. 482.

108. Lissak v. Une Petite Maison, 16 Ind. Bull (N. Y.) 426.

109. Kallis v. Sachs, 16 Ind. Bull. (N. Y.) 207.

110. Firtle v. Kaplan, 15 Ind. Bull. (N. Y.) 64.

111. DeSantis v. Folcaro, 14 Ind. Bull. (N. Y.) 84.

112. Fredello v. Kingsbury Construction Co., 13 Ind. Bull. (N. Y.) 310.

113. Sjoholm v. Hercules Powder Co., 227 Mich. 610; Contra Morgan v. Philadelphia & Reading Coal & Iron Co., 273 Pa. 255.

114. Lippman v. Garfinkle, 16 Ind. Bull. (N. Y.) 296.

115. DeAgostino v. Associated Operating Co., 14 Ind. Bull. (N. Y.) 83.

116. Silver v. Ginsburg News Co. Inc., 16 Ind. Bull. (N. Y.) 297.

117. Strain causing hernia, for example, Jordan v. Decorative Co., 230 N. Y. 522.

TUBERCULOSIS OF THE BREAST

CLARA SEIPPEL WEBSTER, M.D.

Attending Staff, St. Mary's Hospital

TUCSON, ARIZONA

THE subject of this paper appears at first glance to be of academic rather than practical interest, but a review of the literature shows that tuberculosis of the breast occurs frequently enough to deserve careful consideration in the diagnosis of mammary tumors.

Bloodgood found tuberculosis in 6 per cent of benign tumors of the breast. Gatewood reported it present in 1.04 per cent of all breast cases operated upon. At the Mayo Clinic, from 1904 to 1915, ten patients with tuberculosis of the breast represented 0.51 per cent of all mammary conditions admitted to the hospital. Barker tabulated sixty-two cases at the University Hospital at Ann Abor, showing a ratio of one case of tuberculosis to forty-five of malignancy. At St. Bartholomew's Hospital, London, in 1,500 cases of diseases of the breast, 1.5 per cent proved tuberculous.

A little over one hundred years ago (1829) Sir Astley Cooper first described the disease as "scrofulous swelling of the bosom." More than eighty years ago Heyfelder reported the first known case in a male patient. In 1868, Cuneo described another case in a male patient, and he was perhaps the first to isolate the organism from the pus and obtain positive results by animal inoculation.

Diagnosis by histologic examination, so far as is known, was first performed in 1860.

In the year 1881 new impetus was given the study of this disease as a distinct clinical entity by the work of Dubar, whose diagnosis rested not only on finding the tubercle in the tissue, but the bacillus as well.

There are a number of excellent articles on this subject in the literature, that by Maximilian Morgen being one of the most painstaking reviews, covering the period up to 1931. It is upon this paper that I have largely drawn.

There is no unanimity of opinion as to the mode of infection. Some authors believe that in the majority of cases the disease is of hematogenous origin; others that it is a retrograde lymphatic process. In a few rare instances there is no doubt that the entrance of infection was via the ducts, wounds in the skin, or by contiguity of tissue.

Nagaskima performed autopsies on thirty-four patients who died of miliary tuberculosis, in which the breast seemed to be the only organ that was spared; infection by the blood stream probably is not the usual method.

The most logical explanation seems to be a retrograde lymphatic involvement via the axillary, cervical or other lymph glands. There is, somewhere in the body, an unrecognized focus of tuberculosis. We all accept the dictum that "Many are infected with tuberculosis but not all are diseased." In confirmation of the belief in the retrograde lymphatic process, Deaver quoted the French surgeon Duvergey, whose patient, while washing the linens of tuberculous persons, injured one of her fingers and had a suppurating wound for two weeks; eight months later a swelling in the axilla was opened and drained but it did not heal; three months afterward a lump in the breast had to be drained; other sinuses developed; finally the lungs became involved; the Koch bacillus was isolated from the pus exuding from the breast.

In 1914, Deaver made an exhaustive study of seventy-four cases reported in the literature during the previous ten-year period. He found the tubercle bacillus only seven times in stained sections and only

four times in the pus from the necrotic areas.

It is exceedingly difficult to demonstrate the tubercle bacillus in the tissue or the discharge. Gatewood has given this point much study. In every instance in which he was able to isolate the organism it proved to be of the bovine type. We know that the bovine bacillus has a special predilection for lymphoid tissue; this too would strengthen the conviction that the organism enters the breast via the lymphatics rather than the blood stream.

In many cases the axillary glands are enlarged before a mass is recognized in the breast, and fully 50 per cent of the patients have palpable axillary lymph nodes when they consult their physicians.

In several instances carcinoma was found to coexist with tuberculosis, the microscopic study in some cases revealing carcinoma in the breast and tuberculosis in the axillary glands; in others the two pathologic processes were intermingled.

All investigators are satisfied that heredity has practically no part in the problem. Family history of tuberculosis is frequently negative in the primary type.

Trauma, though unquestionably an etiologic factor in a few rare instances, plays but a minor rôle.

Tuberculosis of the breast is, of course, most common in women, and appears most frequently between the ages of 20 and 50. Demme found tuberculosis in the breast of a male infant of six months. Carache, in 1934, reported a case in a woman 74 years of age. Gotthelf of Tucson recently told me of two cases, one in a male patient, both suffering from phthisis, the nature of the breast tumor in each instance being confirmed by histologic examination.

Altogether there are now over 500 cases reported in the literature, at least twenty of them in male patients.

Most of the women were in the childbearing age, and pregnancy and lactation are unquestionably important predisposing factors in the etiology. Out of 366 women with tuberculosis of the breast, 81 (or a little over 20 per cent) were single; of the total 366 women, half had given birth to children.

Clinically, most cases of tuberculosis of the breast belong to the so-called *primary* type—careful search for evidences of tuberculosis elsewhere in the body proves negative. These are often robust, healthy-looking individuals. The *secondary* type is found in patients with a distinct history or evidences of tuberculosis, either active or arrested. Naturally, such patients, as a rule, have only fair general health, or poor.

A lump in the breast is usually the main or only symptom which brings the patient to the physician; she has observed it on an average of six months; sometimes it has existed for years but has only recently become active. Out of 439 cases reviewed by Morgen 132 noticed the lump for six months or less; ninety-seven for more than six months; one for as long as eight years, and another for fourteen years.

Swelling of the axillary glands is present in about half of the cases. Often it is the only abnormality observed by the patient.

Pain is not an early symptom but always sets in when the tumor has grown to sufficient size to stretch the skin, or has broken down.

A lump in the breast may remain quiescent for many years, but once it becomes an active tuberculous process it pursues a progressive course. It has never been known to undergo spontaneous retrogression. Naturally, in the lactating breast it would assume its most fulminant form.

A number of pathologic classifications have been arranged by the different writers on this subject, but about 90 per cent of the tuberculous tumors of the breast can be grouped under three headings:

1. Nodular Variety.

    The breast itself is not necessarily enlarged, but contains one or more firm masses which rarely exceed the size of a hen's egg, and feel like adenomata. This is the most common form.

II. Confluent Variety.

This is a further step in the pathologic process, which assumes a more acute form, and is frequently seen in the lactating breast. The separate nodules have broken down and fused together. Pain develops because of the distention, and the overlying skin becomes tense and red and finally breaks down, releasing a cheesy, purulent discharge. Numerous sinuses may form, each originating in a separate caseating area, or the tracts may communicate with one another.

The cold abscess of Roux belongs to the confluent variety—deeply seated behind the breast tissues where it cannot break through but may gradually burrow its way to the surface.

Fixation of the skin is common in those broken down nodules which lie close to the surface.

Retraction of the nipple ensues when the process is located behind the areola.

III. Scirrhous Variety.

This is most frequently found in elderly women, and is analogous to fibroid phthisis. It resembles scirrhous carcinoma.

The breast is shrunken, distorted and adherent to the chest wall, the nipple retracted, the skin fixed. The pathologic process spreads rapidly to the glands of the axilla, which often break down even though the breast itself remains stationary.

In the gross pathology the nodule often presents a translucent, grayish appearance at the periphery, which fuses into the surrounding tissue; it does not cut with the gritty sensation so typical of malignancy. The center is waxy in appearance and may be necrotic.

Microscopically, it is composed of giant cells of the Langhans type, epithelioid and lymphoid cells, and the usual structures characteristic of tuberculous tissue.

In the 439 cases which Morgen tabulated, fifteen were also carcinomatous; in five adenomata were likewise present; a number showed fibroadenomata. Tuberculosis and carcinoma of the breast may be independent of each other, simultaneous or consecutive. Tuberculosis may be engrafted upon a malignant growth, or carcinoma result from the chronic irritation of the tuberculous process.

The big problem of tuberculosis of the breast lies in its diagnosis, which is exceedingly difficult, especially in its most frequent form—the primary, nodular type—and is usually not made until the time of operation, and much more often only after an operation for supposed carcinoma is completed.

A lump in the breast with palpable axillary glands usually spells carcinoma, and the common treatment by radical mastectomy is indicated. Even if it should prove to be tuberculosis, that too is a disease which threatens life, so that heroic measures are most certainly justified.

If the patient be a woman past 50, perhaps it makes little difference whether a radical or a simple operation has been performed. However, many of the victims of tuberculosis of the breast are women in the twenties and thirties, and it is highly desirable to spare them the mutilation of a radical mastectomy. This may in time result in considerable deformity and even handicap them seriously through partial or total disability of the arm. This point is particularly worthy of emphasis in view of the fact that women will more and more seek medical advice early, when the diagnosis is most difficult.

A positive clinical diagnosis of mammary tuberculosis cannot always be made. There may be a single painless lump with the overlying skin showing the pigskin appearance of carcinoma; an unruptured abscess without inflammatory phenomena, resembling a cyst; discharging sinuses indicating a pyogenic mastitis, broken down carci-

noma or gumma; and the sclerosing type, occurring in the cancer age and much more likely to be diagnosed as malignant.

When a dependable history can be obtained of axillary adenitis preceding by some months the appearance of the lump in the breast, we have a valuable clue to the possible pathology.

In acute or infectious mammary tumors an abscess of the lactating breast is so likely to be regarded as an ordinary pyogenic infection that the true diagnosis is perhaps less frequently made in such cases than in any other form of the disease. As a rule these apparently simple abscesses are opened and drained. The wound heals only to break down later, before the real nature of the infection is determined. Blisnjanska, Lasarevitch and Triousse studied 124 infants born of mothers with open tuberculosis and demonstrated the presence of the tubercle bacillus in the milk of many of these women.

A mammary tumor with sinuses would, of course, arouse our suspicion as to possible tuberculosis, and a biopsy would establish the diagnosis and lead to conservative surgery; for, as one author puts it: "There is practically never any necessity for a radical mastectomy in tuberculosis of the breast."

X-ray of the breast differentiates between the sharply defined cyst-like mass and that which radiates into the surrounding tissue. Lee and Floyd of Philadelphia found in one of their cases of suspected carcinoma the typical diffuse mass which left no doubt as to the malignant nature of the growth. The removed breast showed tubercles. On reviewing the film they recognized a base line, about ½ inch from the chest wall, which, as they state, should have made them think of a non-malignant neoplasm.

In the differential diagnosis, carcinoma outweighs all other conditions in importance. Especially is this true because the nodular variety is the kind most frequently encountered. This is usually operated upon for cancer.

Stuart W. Harrington of the Mayo Clinic emphasizes some excellent points in his paper on the subject: "In the discrete type of tuberculosis of the breast there is often associated adenopathy and fixation of the lesion to the overlying skin; often there is no pain. Some features, however, if present lead to the suspicion that the lesion is tuberculous. Usually mammary tuberculosis afflicts young women and the tumor increases in size more rapidly than a carcinoma. Although a tuberculous tumor of the breast is large when the patient presents herself for examination, metastasis is absent; if such a large growth were malignant, extensive metastasis would be demonstrable by roentgenologic examination. Discharge from the nipple is more commonly a feature of tuberculous than of carcinomatous lesions, and the discharge from a tuberculous lesion is usually composed of pus-like material whereas that from a carcinoma is serous or bloody."

Sarcoma of the breast is exceedingly rare; it grows very rapidly, the cutaneous veins are greatly distended, and pain is intense.

In actinomycosis there would be a history of trauma and the finding of the sulfur granules and the Ray fungus; in tuberculosis the discharge may or may not contain the Koch bacillus, but the tissue would show the tubercle.

Gumma of the breast cannot be diagnosed by a serologic test. McGehee and Schmeisser reported eight cases of tuberculosis of the breast, in 1935, three of which showed positive Wassermann reactions. This did not deter them from operating. One woman with a four plus Wassermann, had noticed a lump in the breast for eight months. The cervical and axillary lymph nodes had been involved but the enlargement disappeared three months previously. A radical operation for carcinoma was performed. The tissue under the microscope presented miliary and conglomerate tubercles.

Wilson reported a case in a woman of 62 who for two years had felt multiple small

tumors in her left breast, which now had attained twice its normal size. The first physician had a biopsy and a Wassermann made, and informed her the growth was syphillitic. Under a course of sixteen arsphenamine treatments she grew steadily worse. The surgeon who saw her next diagnosed malignancy, but, because her condition seemed so hopeless on account of an incompetent heart, he advised against surgery and recommended that she enter a hospital to be made comfortable until the end. Digitalization and rest accomplished unexpected improvement in her general health, so that the mass of supraclavicular glands could be removed. The pathologist reported: "Typical proliferative tuberculous adenitis; no evidence of malignancy or gumma."

After all is said and done tuberculosis of the breast is a baffling problem, and not so uncommon as we are prone to think. Biopsy is the only dependable method by which it can be diagnosed prior to operation; the frozen section, when surgery is undertaken without previous histologic investigation, will determine the nature of the tumor—it will at least disclose the fact that it is non-malignant.

The prognosis is good in the primary type if recognized early and eradicated by proper surgical means. Once tuberculosis of the breast becomes active, however, it usually pursues a more rapid course than carcinoma. Left to itself it advances through extension, particularly into the thoracic viscera, until death ensues. Vandel stated that the average duration of the disease, without intervention, varied from ten months in the primary cases to between eleven and twelve months in the secondary cases.

In the secondary type prognosis as well as treatment depends on the patient's general health and the original infection. With the extensive surgery for the treatment of pulmonary tuberculosis so successfully developed during the past few years, we are unquestionably opening wide the channels of the superficial thoracic lymphatic system, and it would not be surprising if in the future a much larger percentage of the secondary type of tuberculosis of the breast came to our notice.

Amputation of the breast, followed by heliotherapy or roentgenotherapy may make the patient more comfortable in the less hopeful cases.

Treatment, of course, is always surgical in the primary cases and since the latter part of the nineteenth century it has been the generally accepted method. Opening and draining tuberculous abscesses of the breast, scraping or cauterizing fistulous tracts, were methods in vogue before the histologic examination of the tissue was extensively practiced and in the era preceding Koch's discovery of the tubercle bacillus. To employ such measures now is merely to invite disaster.

Some of our leading surgeons recommend preoperative radiation, excision with the cautery knife, and postoperative radiation. Some amputate the diseased breast and dissect out the lymph nodes; others amputate only the breast and treat the glands by x-ray; a few men always do radical operations; most prefer the conservative operation. Rest in the country for some months following surgery is an important feature in the after treatment of some authors; two mention the use of tuberculin ointment and quartz light therapy in early cases, instead of surgery. Efficient treatment by mastectomy, however, is practically universal, and is the only safe and satisfactory method.

SUMMARY

1. Tuberculosis of the breast occurs more frequently than is generally supposed.

2. In a high percentage of cases the patients are young women under 40 years of age.

3. Once it becomes active it pursues a rapidly fatal course.

4. Biopsy offers the only dependable method for accurate diagnosis.

5. Frozen section at the time of operation occasionally shows typical findings of tuberculosis; but it always proves the growth to be non-malignant.

6. Simple mastectomy is the operation of choice, and if performed early makes the prognosis favorable.

REFERENCES

BARKER, H. B. Tuberculosis of mammary gland. *Arch. Surg.*, 13: 435–444 (Sept.) 1926.

BERGER, L., and MANDELBAUM, H. Tuberculosis of the breast. *Ann. Surg.*, 103: 57–66 (Jan.) 1936.

BLISNJANSKA, A. I., LASAREVITSCH, A. I., and TRIOUSSE, M. W. Les bacilles tuberculeux dans le lait de la femme et la destinée des enfants allaités par les mères atteintes de la tuberculose ouverte. *Gynéc. et obst.*, 32: 505–527 (Dec.) 1935.

CHARACHE, H. Tuberculosis of the breast; review of literature and report of cases. *M. Rec.*, 140: 77, 123, 1934.

DICKINSON, A. M. Mammary tuberculosis; report of case in male. *Am. J. Surg.*, 3: 595–597 (Dec.) 1927.

HALL, W. E. B. Tuberculosis of the breast. *Canad. M. A. J.*, 35: 520–525 (Nov.) 1936.

HARPER, H. Y. Tuberculosis of mammary gland. *J. South Carolina M. A.*, 32: 66–69 (March) 1936.

HARRINGTON, S. W. Tuberculosis of the breast. *Surg., Gynec. & Obst.*, 63: 797–798 (Dec.) 1936.

JESSEL, G. Tuberculosis of the mammary gland, *Brit. M. J.*, 1: 1023 (June) 1931.

LEE, W. E., and FLOYD, W. R. Tuberculosis of the breast. *Ann. Surg.*, 99: 753–759 (May) 1934.

MAHONEY, L. E. Primary tuberculosis of the breast. *Am. J. Surg.*, 18: 97–98 (Oct.) 1932.

MARTIN, W. F. Tuberculosis of mammary gland. *South Med. & Surg.*, 96: 67–69 (Feb.) 1934.

MORGEN, M. Tuberculosis of the breast. *Surg., Gynec. & Obst.*, 53: 593–607 (Nov.) 1931.

McGEHEE, J. L., and SCHMEISSER, H. L. Tuberculosis of the breast. *Am. J. Surg.*, 28: 461–488 (May) 1935.

SWAN, R. H. J., and FRY, H. J. B. Tuberculosis of the male breast. *Brit. J. Surg.*, 14: 234–242 (Oct.) 1926.

VANDEL, D. T. Tuberculosis of mammary gland; case history study. *J. Missouri M. A.*, 24: 260–263 (June) 1927.

WILSON, G. E. Tuberculosis of the breast. *J. A. M. A.*, 102: 128–129 (Jan. 13) 1934.

MERTHIOLATE: A TISSUE PRESERVATIVE AND ANTISEPTIC

Gerald Brown O'Connor, M.D., F.A.C.S.

Plastic Surgeon, St. Mary's Hospital

SAN FRANCISCO, CALIFORNIA

I HAVE found clinically and experimentally in the past five years that merthiolate (Lilly), both the tincture and the aqueous solution, is a most efficient and highly satisfactory preservative and antiseptic in reconstruction surgery. I use this product practically exclusively in preoperative preparation in all types of skin grafts and other plastic problems. The antiseptic qualities of merthiolate,[1] although of secondary consideration in this report, strengthens its value as a preservative agent. The preservative action has been studied along other lines,[2] but I am particularly concerned here with the use of merthiolate in the preservation of what I call "refrigerated cartilage isografts."

These grafts are used to reconstruct all facial appendages and repair contour defects of the forehead, nose, ear, chin, eyesocket and other parts of the facial framework. The cartilage, mainly rib, is obtained under sterile or unsterile conditions and is stripped of its perichondrium. It is then thoroughly washed with ordinary tap water to remove any blood which may have collected. It is then placed in a sterile glass container and covered with a solution of one part aqueous merthiolate (1:1000) to four parts of sterile normal saline. The solution should completely cover all of the cartilage by an excess of at least 1 inch. The container is covered and placed in the refrigerator. It is taken out only when the solution has to be changed or cultured, or the refrigerated cartilage is to be used for grafting purposes. When new cartilage material is prepared, the solution is drained off and changed twice a week for two weeks and once a week thereafter. New cartilage material

should not be added to the sterile original container unless it has undergone the "merthiosaline" treatment for at least one week and two cultures of the second preservative solution are negative. I have made it a routine to obtain a culture from the "merthiosaline" solution every time a piece of refrigerated cartilage is to be used for reconstruction. There has never been a positive culture return from the preservative solution in over 375 cases.

I have used refrigerated cartilage isografts as early as twenty-four hours and as late as two years after they have been placed in the "merthiosaline," but I strongly advise that this type of graft not be used until it has been refrigerated in the solution for at least one week and until negative cultures of the solution are obtained by the operator. Refrigerated cartilage isografts that have been preserved over six months are just as efficient as fresh cartilage. The long waiting period, although not advisable, does not materially affect the cartilage as grafts.

Macroscopically the fresh and older cartilage is identical, except that some of the older material is slightly more pliable than when first obtained. Microscopically small vacuoles occasionally were noted in the sectioned cartilage. Refrigerated cartilage isografts, when transplanted, bend occasionally, but with much less frequency than autografts. This is probably due to the fact that when the perichondrium is removed and the cartilage is placed in the "merthiosaline" it assumes the shape that the internal strains and stresses of the cartilage cells dictate. Grafts can then be sculptured from this cartilage of the required shape to fit the existing defect. Cartilage isografts have been accurately

sculptured and then placed in the "merthiosaline," which sterilized and preserved the graft until it was ready for use.

In 375 transplants, local infection developed in six cases, mainly in grafts for reconstruction of the nasal bridge or tip. These infections responded to local treatment. All the grafts remained in situ except for any portion which became exposed; the exposed portion of the graft was gradually digested or was surgically excised to speed up wound closure. The latter was the usual procedure as it materially shortened the period of postoperative care.

If a refrigerated cartilage isograft becomes infected there are several alternative procedures possible. The whole cartilage graft may be removed immediately, after which the infection subsides rapidly and the wound heals in a short time. If good dependent drainage is established the graft can be left in situ and it will survive, but healing is delayed. To speed recovery where nasal bridge transplants became contaminated, I established intranasal or columellar drainage points and grafts were irrigated with "merthiosaline" introduced through the nasal skin into the graft bed by hypodermic syringes. This should not be done under pressure, and only when through and through drainage has been established. The solution merely acts as an irrigant, injected into the graft cavity and not the skin or subcutaneous tissue.

One total graft was removed; it was taken out, due to streptococcus infection, through its point of introduction, forty-eight hours after being placed under the skin of the nasal bridge. The symptoms subsided under local treatment as soon as the graft was removed and adequate drainage provided. This infected graft was thoroughly washed in saline solution, refrigerated in the "merthiosaline" and kept for six months. It was then felt that the site to be grafted had recovered from its infection and the original graft was reintroduced. Healing occurred per primam. Infected cartilage can be removed, re-

sterilized, preserved in "merthiosaline" and used again.

After a five year waiting period all but seven of the transplanted refrigerated cartilage isografts have retained their original size and identity. Parts of six grafts became exposed and one small nasal bridge graft was entirely absorbed.

The one graft that completely absorbed was $7/8$ inch long, $2/8$ inch high and $3/8$ inch wide. It was used to build up a luetic nose which was noticeably lacking in skin covering and mucosal lining. The graft was unfavorably situated under tension and an opening occurred between it and the nasal cavity. The continued mild infection plus the exposure of the graft eventually caused its disappearance. An additional refrigerated cartilage isograft was inserted after a six month waiting period and has remained in situ for three years.

My methods, technique and results have been presented to others in communications, and many have used this type of graft with success.

COMMENTS

1. After five years' experience I feel that merthiolate has proved a highly satisfactory antiseptic and preservative for cartilage isografts.

2. Refrigerated cartilage isografts have been successfully preserved and utilized as contour building material as late as two years after preservation.

3. The cartilage grafts are not materially changed by their preservation and maintain their identity indefinitely after grafting.

4. Refrigerated cartilage isografts are most efficient, highly satisfactory and economical for the patient.

5. In my work refrigerated cartilage isografts have entirely replaced the autogenous cartilage graft which previous to this experimental work had been our choice of grafts for certain types of contour restoration.

I express my sincere appreciation to Dr. A. Moody and Dr. S. Leland of San Francisco for the cooperation and assistance they have rendered in helping to solve the many problems that confronted me in this work.

REFERENCES

1. (a) Buchsbaum, R., and Bloom, W. Relative toxicity of antiseptics and tissues in culture. *Proc. Soc. Exper. Biol. & Med.*, 28: 1060–1064, 1931.
   (b) Marks, H. H., Powell, H. M., and Jamieson, W. A. Merthiolate as a skin sterilizing agent. *J. Lab. & Clin. Med.*, 17: 443–449, 1932.
   (c) Marshall, M. S. Methiolate—a new antiseptic. *California & West. Med.*, 35: 43–44, 1931.
   (d) Powell, H. M., and Jamieson, W. A. Merthiolate as a germicide. *Am. J. Hyg.*, 13: 296–310, 1931.
   (e) Powell, H. M., and Jamieson, W. A. Comparative studies on merthiolate with reference to laboratory evaluation and human tissue antisepsis. *Proc. Ind. Acad. Sc.* 41: 249–256, 1931.
   (f) Powell, H. M., Jamieson, W. A., and Kempf, G. F. The healing properties of merthiolate. *Am. J. Hyg.*, 15: 292–297, 1932.
2. (a) Morgan, C., Jamieson, W. A., and Powell, H. M. Merthiolate as a preservative for biological products. *Am. J. Immunol.*, 25: 121–126 (Aug.) 1933.
   (b) Powell, H. M., Jamieson, W. A., and Jones, F. G. Merthiolate as a preservative for biological products. *J. Immunol.*, 24: 185–192 (March) 1933.

The modern tendency is to look upon non-tuberculous renal infection as a single pathologic entity presenting various types, dependent on the location and bacterial type and cause of the original lesion, on the course of its evolution and on the extent of tissue destruction that has been sustained.

From—"Manual of Urology" by R. M. LeComte (Williams & Wilkins).

FAMILIAL OSTEOPOIKILOSIS*

A. HINSON, M.D.

Associate Surgeon, Davis Hospital

STATESVILLE, NORTH CAROLINA

Definition. A rare, familial disease characterized by multiple areas of condensation in the epiphyses and metaphyses of bones. These areas vary in size and shape. They may be spherical, elongated, lenticular, or striated in character. The striated or ray type never extends into the ephiphyses proper but seems to stop abruptly at the epiphyseal line.

Synonyms. This disease has been wrongly called osteosclerosis fragilis generalisata. Osteitis condensans generalisata has been used, but the disease is evidently not of an inflammatory nature. It was called osteopoecilia by Chabaneix and Dessane. Newcomet thought the disease was a multiple enostosis and called it spotted bones. Wachtel named it osteopathia condensans disseminata.

Etiology. The exact cause of osteopolkilosis is unknown. A number of diseases have been found coexisting with this condition and have been thought by some to play a part in its origin. Svad[1] reports a number of cases with a concurrent diabetes. Shinz, Baensch and Friedl[2] think that it is a maldevelopment probably arising by a mutation and recurring recessively. Konig,[3] Wachtel,[4] Steenhuis,[5] and Moreau[6] advanced the theory that it was due to emboli in the terminal arteries. Wilcox[7] thought that it was due to more or less uniform disturbance in proliferation and ossification of bone forming cartilage during the period of development. Newcomet[8] thought the condition was due to multiple enostosis extending into the bone from the periosteum. Ledoux-Lebard, Chabaneix and Dessane[9] found a history of typhoid in their cases. Schmorl[10] described the condition as being hereditary, possibly related to a parathyroid disturbance. There is no conclusive evidence that shows any relation between the condition and any infectious disease as no one disease has been present in all cases. Nichols and Shifflet[11] thought it was due to an abnormal relationship between osteoclasts and osteoblasts. Voorhoeve[12] thought that it was related to chondrodysplasia.

There is some evidence that the condition may be related to the arterial system as the general distribution follows that of hematogenous tuberculosis in the bone. However, this is far from proved. The fact that it is scattered throughout the metaphyses and epiphyses, with no direct relation to the epiphyseal line, would tend to disprove any connection with cartilaginous bone formation. The idea of these areas being enostosic would be disproved by the fact that they were not in contact with the cortical bone as they would be were this true. In one of my cases definite cystic areas were found in the cortex of the metatarsal bones with a very condensed area immediately surrounding. These areas bulged externally and internally from the cortex. This condition has not been previously noted in osteopoecilia. There can be no doubt as to the cortical location of these areas, and furthermore they are about the size of cancellous bone lesions found elsewhere.

*Plates and films by W. L. Gerald.

Age, Incidence and Distribution. The youngest patient reported is in a case of Heilborn,[13] in a fetus of 4 months. An

Fig. 1. Pelvis and hips with disseminated lesions of osteopoikilosis.

occasional case has been found in infancy and childhood, but most instances are found between the ages of 15 and 60.

To date about seventy-two cases have been reported in the literature, the greater number in Europe. About ten articles have appeared in the United States. The present report covers seven cases in one family.

These lesions have been reported in practically every bone in the body with the exception of the skull; in one instance they were found in the occipital bone. In a large number of cases they are absent from the pelvis and vertebrae. They seem to have a predilection for the epiphyses and metaphyses of the long bones, being most frequently found in the small bones of the wrist, the hands and the feet. The small, rounded lesions are generally found in the epiphyses but may be found in the metaphyses. The linear lesions are practically never found in the epiphyses and are found in the cancellous bone. Changes in the external lines of the bone have not been noted before.

The occurrence of the bony lesions in eight members of one family certainly goes a long way to prove the hereditary tendency of the disease. It is expecting too much of coincidence to have such a rare condition show up in so many cases in the same family.

The disease was first reported by Stiede[14] in 1905. The next case reported was that by Albers-Schonberg[15] in 1915; next, Ledoux-Lebard[9] in 1913, Moreau[6] in 1916, Wachtel[4] in 1923, Richarz[16] in 1922, Schele[17] in 1922, Voorhoeve[12] in 1924, Steenhuis[5] in 1926, Buschke-Ollendorf[18] in 1928, Van Dorp and Beucker[19] in 1928, Gluch[20] in 1929. The first case reported in this country was that by Newcomet[8] in 1929. Amundsen[21] reported a case in 1930, Guelliars and Mollaret[22] in 1930, Awalischwili[23] in 1930, Konig[3] in 1930, Schmorl[10] in 1931, Reiser[24] in 1930, Haack[25] in 1931, Van Pee[26] in 1931, Wilcox[7] in 1931, Windholz[27] in 1932, Bloom[28] in 1933, Holly[29] in 1936, Nichols and Shifflet[11] in 1934, Bach[30] in 1931, Mascherpa[31] in 1931, Nather[32] in 1936, Hirsch[33] in 1935, Sutherland[34] in 1935, Kadrnka and Hirlemall[35] in 1933, and Jeter and McGeehee[36] in 1933.

Voorhoeve[12] found the condition occurring in the father, brother and sister; Wilcox[7] in the father and daughter. Bloom[28]

FIG. 2. Large number of lesions, elongated, circular and lenticular, in the femur, patella and tibia.

reported an isolated spot in the sister of his patient.

Symptoms. Osteopoikilosis is apparently asymptomatic, generally being found during roentgenographic study for some other lesion. In most instances the discovery of the disease in one region of the body leads to further study of the entire body. In some instances, as in my cases, the patients have complained of indefinite pains in the back and thighs, and in the two cases of the ray type hydrarthrosis of the knee had been present. Windholz[27] and others report the presence of a disseminated lenticular dermatofibrosis in their case. This is characterized by slightly elevated papules in various parts of the body usually close to each other. They are oblong or oval-shaped, about the size of a lentil, and may be longitudinal stripes.

There is a fibromatous thickening of the cutis without tumor formation. The elastic fibers are usually well preserved within

FIG. 3. Cortical nature of lesions in third metatarsal bones.

these areas. Some authors believe that the bony and skin changes are both a part of a constitutional anomaly of connective tissue. Others believe it to be similar to scleroderma.

The occurrence of osteopoikilosis along with congenital syphilis, tuberculosis, and various bony changes of other types has been mentioned. The lesions are usually bilateral in character but are often missing from some bones. The nodules are very dense in comparison with surrounding trabeculae and vary in size from 1 to 4 cm. Some authors have followed them over a period of years and have noted no change in the size and number of islands.[8] Holly,[29] however, states that in four cases which he followed for five years a definite change occurred in the islands of condensation. Some entirely disappeared and many of the lesions became enlarged, especially those in children. In some instances he noticed fusion of two or more areas. Some became

smaller and there was fading of some of the larger spots. Some of the round nodules became elongated and many coalesced. While the lesions are usually distinct from the surrounding bone, some gradually fade away into what appears to be normal spongiosa.

There is no increased fragility in these bones and though a number of cases have been followed carefully, no constant changes have been noted in the blood counts or blood chemistry. In a few cases there were changes in the calcium and phosphorus content of the blood, but this was not constant enough to be significant.

Differential Diagnosis. Osteopoikilosis must be differentiated from carcinoma, sarcoma, syphilis, osteomyelitis, tuberculosis and typhoid bacillus infection.

In bone forming carcinomatous metastasis the differential diagnosis is hard unless the entire skeleton is examined. However, a careful search for a primary lesion and x-ray examination of the entire skeleton will generally reveal the difference.

The changes seen in sarcoma, syphilis, osteomyelitis, typhoid and tuberculosis are quite distinct and should give no trouble in differentiation. The clear, distinct nature of the nodules and their distribution should result in early diagnosis if the condition is kept in mind. Some difficulty may be encountered in cases of osteopoikilosis concomitant with these diseases.

Melorheostosis of Leri must also be differentiated. This usually is diagnosed by the hyperostotic processes always limited to one extremity. The direction of growth here is characterized by considerable striated density of the bone but extends from the flat bone of the root of the extremity to the terminal phalanges. This gives rise to clinical symptoms such as pain and limitation of motion.

Pathology. Two cases have come to necropsy study. Schmorl[10] in 1931 performed an autopsy on a patient with osteopoikilosis. Death was due to widespread osteomyelitis with amyloidosis and destructive luxations and contractions of many joints. Hemolytic streptococci and proteus organisms were found in the purulent exudate. This patient had exten-

FIG. 4. Multiple lesions around the shoulder joint.

sive calculus formation in both suprarenals and in the wall of an abscess around the left kidney. *X*-ray examination showed the disseminated osteopoikilie in all parts of the skeleton except the clavicle, vertebrae and skull.

On microscopic examination the bone foci, which on x-ray and visual examination appeared to be made up of compact bone, appeared dissimilar to the tissues corresponding to the compacta of the shaft bones. These foci consisted of spongy bone similar to the spongiosa. The bony trabeculae were thicker than the normal spongiosa trabeculae and plates otherwise present in that location. The individual trabeculae, of which the foci were composed, lay very close together and were thicker; therefore on inspection of the foci the impression of a compact bone island was obtained. On serial section the foci faded into the normal spongiosa and were connected with it in such a way that the spongiosa trabeculae were adjusted to their surface without a demonstrable sharp boundary.

Normal bony trabeculae may be found adjoining the surface of the foci. Besides bony trabeculae with a lamellar structure newly developed connective tissue bone. Schmorl[10] stated that no reliable verdict as to the genesis of these trabeculae could
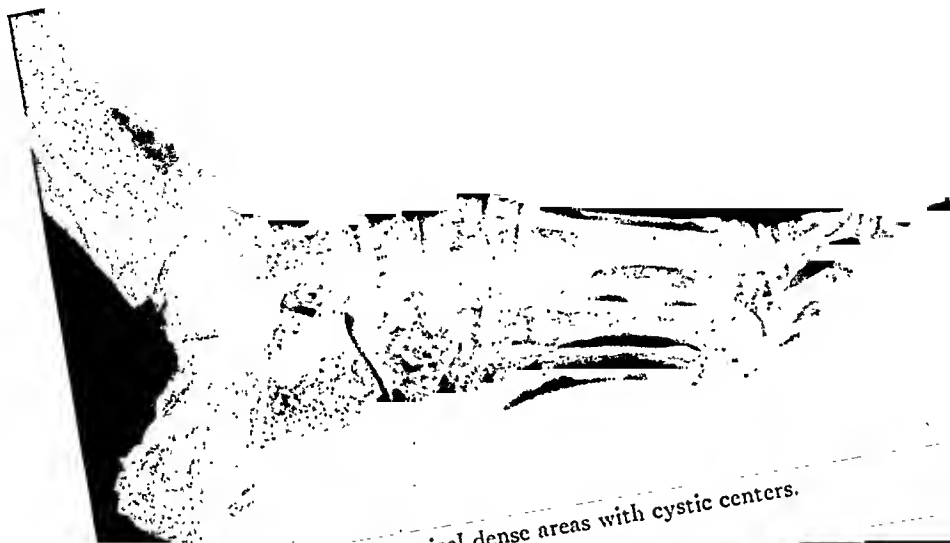
FIG. 5. Cortical dense areas with cystic centers.

FIG. 6. Epiphyseal type of lesions in the radius and ulna with many islands in the carpals and metacarpal bones.

there are others, especially thinner ones, which are composed of fine, fibrillar bone tissue. The bone corpuscles contained within are often rather large as are found in

be derived on the basis of microscopic examination. He felt that the theory of enchondral bone growth could not explain them as they were not found in the areas

where this type of growth generally takes place. No cartilaginous remnants could be found anywhere in the tissue examined.

Fig. 7. Typical appearance of knee joints.

The pathology of the case reported by Windholz[27] was that of thickened trabeculae in the vicinity of the bone island. The focus consisted of closely arranged trabeculae with no sharp line of demarcation between normal bone and island. Fine vessels were found entering into formation between spaces of separate bundles of spongiosa. Small medullary spaces were found between trabeculae. The trabeculae were not parallel to the surface of formation but were radiating and had a rather perpendicular direction. A rather important observation here was the absence of osteoblasts. Osteoclasts were noted. No Haversian systems were present. There was no difference between the compacta and the normal bone in either case here, nor any signs of inflammatory reaction.

DISCUSSION

Osteopoikilosis is a rare familial bony change. Beyond this it is hard to make any definite statement. The literature is full of contradictory conclusions obviously based on an inadequate number of cases. The condition is apparently asymptomatic. It has occurred with periostitis, osteomas, chondromas, and syphilis, both congenital and acquired. It has been reported with tuberculosis, diabetes and disseminated lenticular dermatofibrosis. The diagnosis is easy if it is kept in mind. The pathology is apparently a localized overgrowth of the normal bony structure. Why it assumes the patterns seen is unknown. Possibly it is due to some congenital anomaly of the terminal arteries activated by glandular dyscrasia, an "acromegaly" of discrete islands of bone. Holly's[29] cases disprove the idea that the islands do not change in shape or size. My first case shows definite islands in the cortical bone with bulging of the normally smooth line of the shaft.

The ray type has not been examined pathologically. It is such a rare, bizarre condition that we should see further cases before stating that it is osteopoikilosis.

It is only by reporting these cases as they occur that definite conclusions as to their significance may be drawn.

CASE REPORTS

CASE I. G. R., age 38, white, single, was admitted to the hospital on December 29, 1937 with a complaint of "rheumatic" pains along the right sciatic nerve. She had had previous attacks of sciatica ten years before. She was in bed for six weeks before admission and could not walk without extreme pain along the course of the right sciatic nerve.

Family history showed no evidence of tuberculosis, cancer, hemophilia or mental disease. The only fracture that occurred in the immediate family was in the mother who was 65 years old. This had occurred forty years before and had healed normally.

The patient had had measles, influenza, rheumatism, whooping cough, tonsillitis and grippe. Her tonsils were removed in November, 1937. She complained of an occasional headache and had a poor appetite.

Menstruation had begun at the age of 12, regularly occurred every twenty-eight days, for five days and was not painful.

General examination revealed a thin, white, undernourished female, height 5 feet 8 inches, weight 118 pounds, pulse rate 90, blood pressure 135/80. Behavior was normal. Careful examination of the eyes, ears, nose and throat was negative except for the absent tonsils. The thyroid gland was not enlarged. The breasts were slightly atrophic. The cardiovascular and respiratory systems were apparently normal. The abdomen was flat and contained no masses. No herniae were present. Pelvic examination showed an intact hymen which prevented a vaginal examination. Palpation by rectum showed the uterus to be in normal position with no adnexal pathology.

The patient complained of tenderness over the course of the sciatic nerve from where it left the pelvis on the right down to the popliteal space. Lesage's sign was positive.

The urine showed a specific gravity of 1.014, was amber in color and acid in reaction. Albumin, two plus, was found. Microscopic examination showed about 80 pus cells. Red blood count was 4,250,000, hemoglobin 88 per cent, white blood count 7,500, with polys 70 per cent, lymphocytes 24 per cent, and monocytes 6 per cent. Sedimentation rate was over three hours. The blood sugar was 116, blood calcium 8.7, uric acid 3.1. The Kahn test was negative. Examination of a vaginal smear was negative for gonococcus and trichomonas vaginalis.

X-ray of the lumbar spine and pelvis showed multiple areas of condensation of the spine, hips and pelvic bones. These areas were from 1 to 3 mm. in diameter and were closely divided from the surrounding apparently normal bones. They did not occur in the cortex, but in the spinous process and portions of the bodies of the vertebrae. The largest number occurred near the joint surfaces. There were several elongated areas in the metaphyses of the femora.

These findings led to a complete study of the skeletal structure. The same disseminated areas were found in the shoulders, knees, ankles and feet. The skull was negative. In the metatarsals definite areas were found in the cortex of the bone. Some were smooth spheroids projecting outward and inward from the normal cortex. The distinct, smooth outline would tend to disprove an inflammatory origin. Some of these dense areas in the cortex had irregularly cylindrical clear areas for centers, with dense island bone outside. These areas were not much larger than the normal islands of osteopoecilia.

CASE II. M. G. R., mother of the first patient, age 65, had had nine children. She had always been in good health and had had no serious illness.

X-ray showed circular nodules in the small bones of the wrist, metacarpals and phalanges.

CASE III. F. M. T., sister of the first patient, age 39 had no history of illness or broken bone.

X-ray of the wrist showed a moderate involvement with the circular type of lesion of the lower end of the radius and ulna, the small bones of the wrist, the metacarpals and the phalangeal bones.

CASE IV. R. G. R., a brother of Case I, age 44, had a negative past history except for meningitis in 1919 at which time he lost three toes, apparently from a gangrenous process. No other serious illness had occurred.

X-ray of the pelvis, lower spine and hips showed the typical disseminated character of the osteopoikilosis, particularly marked in the hips, the neck and trochanters of the femora and the pubic bones. X-ray of the wrists, hands and feet showed the same process in practically all the bones.

CASE V. C. R., another brother, age 22, had had a broken finger in a football game several years before, and an appendectomy.

X-ray of the wrist showed very large, discrete condensations of the lower ends of the radius

and ulna, the small bones of the wrist, the metacarpals and phalangeal bones.

CASE VI. V. W., a sister, age 30, had had an appendectomy eight years before, but no other serious illness.

X-ray of the pelvis, hips, lumbar spine, shoulders and feet showed the typical circular, lenticular and elongated lesions of osteopoikilosis, especially marked in the epiphyses.

CASE VII. L. R., a sister, age 40, had had no serious illness.

X-ray of the wrists and hands showed one small area of osteopiokilosis in the lower end of the radius on the left.

CASE VIII. N. M. R., a niece of the first patient, age 14, had a negative past history except for tonsillectomy.

X-ray of the wrist, hand, pelvis, hips, ankle and foot showed many disseminated lesions of osteopoikilosis in all these areas.

Two nephews and another sister of Case I were examined, but proved negative for osteopiokilosis.

REFERENCES

1. SVAD, V. A propos de l'osteopoecilie héréditaire. *J. de radiol. et d'électrol.*, 16: 405–415, 1932.
2. SHINZ, H. R., BAENSCH, W. and FRIEDL, E. Osteopoikilie, 3rd ed. George Thieme, Leipzig, 1: 289.
3. KONIG, E. Osteopoikilie. *Fortschr. a. d. Geb. d. Röntgenstrablen*, 42: 406–407, 1930.
4. WACHTEL, H. A case of disseminated condensing osteopathy. *Fortschr. a. d. Geb. d. Röntgenstrablen*, 27: 624–626, 1921.
5. STEENHUIS, D. J. About a special case of ostitis condensans disseminata. *Acta radiol.*, 5: 373–374, 1926.
6. MOREAU, L. Sur un nouveau cas d'ostite condensante generalisee (osteopoecilia). *J. de radiol. et d'électrol.*, 3: 318–319, 1917.
7. WILCOX, L. R. Osteopoikilosis (disseminated condensing osteopathy). *Am. J. Roentgenol.*, 27: 580, 1932.
8. NEWCOMET, W. S. Spotted bones. *Am. J. Roentgenol.*, 22: 460, 1929.
9. LEDOUX-LEBARD, R., CHABANEIX and DESSANE. New form of generalized condensing ostitis without clinical symptoms. *J. de radiol. et d'électrol.*, 2: 3, 1916.
10. SCHMORL, G. Anatomic findings in a case of osteopoikilosis. *Fortschr. a. d. Geb. d. Röntgenstrablen*, 44: 1–8, 1931.
11. NICHOLS, B. H., and SHIFFLET, E. L. Osteopoikilie. *Am. J. Roentgenol.*, July, 1934.

12. VOORHOEVE, N. A hitherto undescribed picture of abnormality of the skeleton. *Acta radiol.*, 15: 407, 1924.
13. HEILBORN. Quoted by Hirsch.[33]
14. STIEDE, A. Circumscribed densening of the bone in the region of the substantia spongiosa as found roentgenologically. *Beitr. z. klin. Chir.*, 45: 700–703, 1905.
15. ALBERS-SCHONBERG, H. E. A rare unknown structural anomaly of the skeleton. *Fortschr. a. d. Geb. d. Röntgenstrablen.*, 23: 174–175, 1915.
16. RICHARZ. Multiple Kompaktaherde in der Knochenspongiosa. *Fortschr. a. d. Geb. d. Röntgenstrablen*, 28: 86–87, 1921–1922.
17. SCHELE, A. A case of a rare skeleton anomaly. *Acta radiol.*, 1: 536–539, 1921–1922.
18. BUSCHKE, A., and OLLENDORF, H. A case of dermatofibrosis lenticularis desseminata and osteopathia condensans disseminata. *Dermat. Wcbnscbr.*, 83: 257–262, 1928.
19. VAN DORP, D. B., and BEUCKER, A. Demonstration of x-ray photographs of a case of osteopathia condensans disseminata in a patient with abdominal tuberculosis. *Nederl. tijdschr. v. geneesk.*, 72: 1012–1013, 1928.
20. GLUCH, G. Ueber einen Fall von Osteopoikilie. *Röntgenpraxis*, 1: 505–507, 1929.
21. AMUNDSEN, P. A case of disseminated condensing ostitis. *Acta radiol.*, 11: 120, 1930.
22. GUELLIARS, G., and MOLLARET, P. Achondroplasia with osteopoecilia and vitiligo. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 54: 214, 1930.
23. AWALISCHWILI, G. Incidence of osteopoikilia. *Röntgenpraxis*, 2: 831–833, 1930.
24. REISER, E. Osteopoikilie. *Fortschr. a. d. Geb. d. Röntgenstrablen*, 43: 808, 1931.
25. HAACK, E. On osteopoikilia. *Röntgenpraxis*, 3: 611–613, 1931.
26. VAN PEE. Osteopoikiliosis and condensing osteitis. *Liège-méd.*, 24: 937–946, 1931.
27. WINDHOLZ, F. Familial osteopoikilosis and disseminated lenticular dermatofibrosis. *Fortschr. a. d. Geb. d. Röntgenstrablen*, 45: 566, 1932.
28. BLOOM, A. R. Osteopoecilia. *Am. J. Surg.*, 22: 239, 1933.
29. HOLLY, L. E. Osteopoikilosis; five year study. *Am. J. Roentgenol.*, 36: 512, 1936.
30. BACH. Osteopoikilosis. *Zentralbl. f. Chir.*, 58: 2218, 1931.
31. MASCHERPA, F. Osteopoikilosis. A new case of striated, disseminated condensing osteopathy. *Radiol. med.*, 18: 1014, 1931.
32. NATHER, F. B. Osteopoikilosis, report of four cases. *Am. J. Roentgenol.*, 35: 495, 1936.
33. HIRSCH, I. S. Osteopoikilosis. *Radiology*, 25: 349, 1935.
34. SUTHERLAND, C. G. Osteopoikilosis. *Radiology*, 25: 470, 1935.
35. SILVIJE, K. and ARNOLD, H. Familial osteopoikilosis and congenital syphilis. *Rev. d'orthop.*, 20: 29.
36. JETER, HUGH, and McGEHEE, C. L. Osteopoikilosis; case report. *J. Bone & Joint Surg.*, 15: 990, 1933.

PLICATION OF THE SMALL INTESTINE

SECOND REPORT

Thomas B. Noble, Jr., m.d.

Visiting Surgeon, Methodist and St. Vincent Hospitals

INDIANAPOLIS, INDIANA

IN The American Journal of Surgery of January 1937, under the title "Plication of the Small Intestine as Prophylaxis Against Adhesions" the first description of the uses of plication appeared and the method was described in its simplest form. It was believed at the time that understanding of plication in a single unit would naturally suggest its application in a more complicated form. Many contacts resulting from the publication of the above article have proved the correctness of this belief. A description of a more complex use of plication has seemed warranted therefore, particularly with reference to its use in a serious type of appendicitis.

When the acutely inflamed appendix lies low and in the pelvis surrounded by loops of small bowel, rupture means widespread peritonitis, sepsis, abscesses in mesenteric folds, multiple areas of constricted gut, intestinal obstruction, and a high percentage of fatality. Distention is met early and is perhaps the most active factor in an unfavorable outcome. Enterostomy has seemed the only hope; but the underlying pathologic state composed of constricted gut and thrombophlebitis of mesenteric vessels, abscesses in mesenteric folds, and infection under tension have all too often meant death from sepsis even though distention has been relieved through enterostomy.

For a number of years we have followed a plan more aggressive than that of so-called symptomatic treatment, which in this case consists of following after the developing and unimpeded pathologic state. In these cases of ruptured appendix with widespread peritonitis, we first do an appendectomy through a small McBurney incision leaving a rubber tube drain—not a Penrose—into the bottom of the pelvis. This may be done under novocaine or any gas-oxygen combination as fits the needs of the case. Postoperatively, as little morphine as possible is given, because of its depressing effect upon metabolism and probably upon liver function.

Surgical pituitrin is given, one ampule hourly or as needed, to prevent distention. This is instituted immediately after the appendectomy. Any sudden fall of temperature means that tolerance has been passed and that the pituitrin should be reduced or skipped for two or more hours, or replaced with pitressin for a short time. Intravenous glucose and liquid diet supply all nutrition until the outcome is decided by second operation, recovery, or failure. If distention can be kept down for two or three weeks, the outcome becomes more certain. Immunity must be developed and an accurate measure of this may be had from the clinical appearance and from the disappearance of nausea and the beginning of hunger. Observation has taught us that no accurate picture of the extent of the trouble at this time can be established.

Second operation has been done in our cases from two to four weeks after appendectomy, and is necessitated by persisting tendency toward distention, scattered tenderness, perhaps fever, and an increased percentage of polymorphonuclears. At this time in our cases there have been found multiple abscesses in mesenteric folds ranging in size from a minute spot to those containing as much as 4 ounces of thick pus. Rare cases have hidden as much as a pint behind the sigmoid, and secondary drainage is indicated here.

FIG. 1. Appearance of distended loops of ileum, noticeably congested on opening of abdomen. Gaseous distention after twenty-nine days of pituitrin.

FIG. 2. One region of intestine densely adherent to floor of pelvis. Denuded peritoneum, plastic lymph, constricted bowel, marked local inflammation. In the mesenteric fold lay an ounce of thick pus, other areas containing other abscess areas up to size of a baseball.

Noble—Plication

576

FIG. 3. Illustrating the adherence of a loop to the root of the mesentery with consequent increasing constriction and obstruction. Gas has rapidly filled the more collapsed bowel as partial liberation has occurred in presenting the region to the camera. It is this adherence to the root of the mesentery, illustrated here, which must be prevented from recurring if the patient is to remain well.

FIG. 4. After complete release the length of the mesentery permits the suturing of the intestine to be done extra-abdominally. The lines of suture in making the mesenteric approximation of plication are marked out by the Allis clamps.

Fig. 5. A single loop of plication completed, covering a mesenteric abscess and rough area of intestinal peritoneum.

Fig. 6 Five adjacent and continuous loops of plication with one wing anastomosis because of an annular constriction. Here is shown a most severe degree of destruction of peritoneum of all surfaces of the bowel. No manner of plication will cover all surfaces. Adhesion to the root of the mesentery of some loop of intestine as shown in Figure 3 is prevented by plication, and obstruction will not appear later. Omentum if present will substitute for the missing external surface of the peritoneum of the intestine, preventing fixation to the abdominal wall. If not present, as in Figure 10, the fixation to the lower parietal peritoneum of such denuded external surface of the intestine produces no degree of obstruction or symptoms referable to intestinal function, if plication has been complete, surprising though it may seem. The patient here shown has made a completely satisfactory recovery without drainage or infection, and remained free of symptoms and has returned to his gainful occupation.
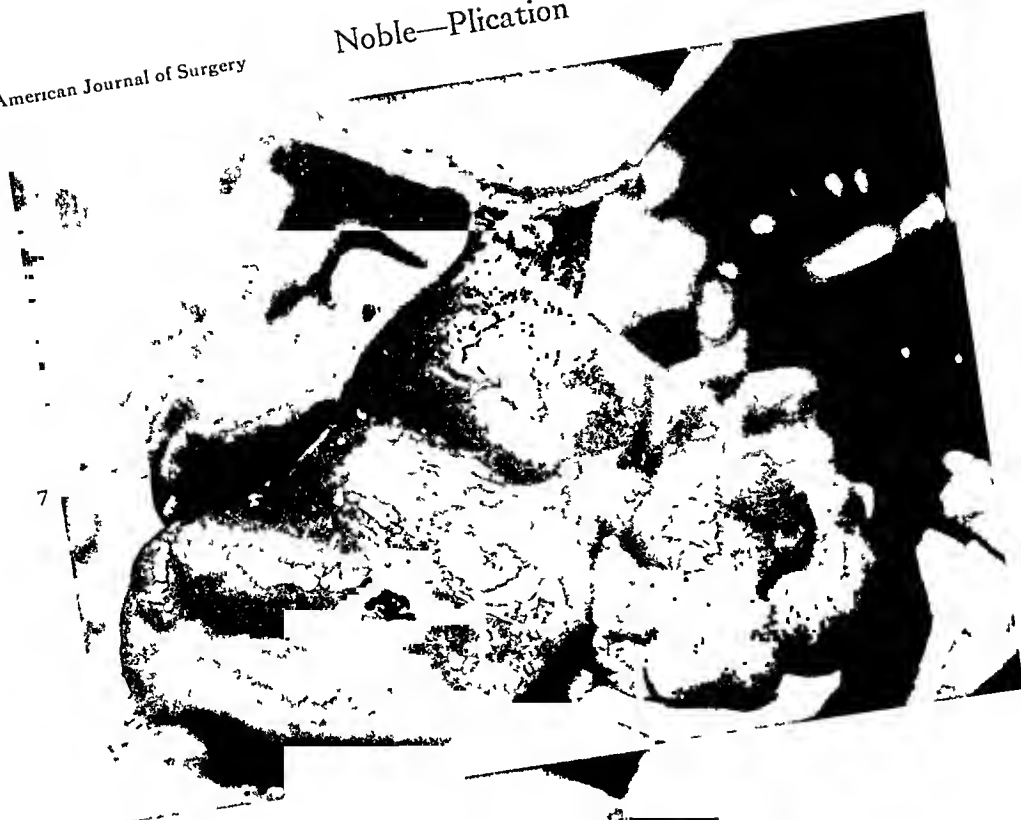
FIG. 7. A case of long standing partial obstruction following resection of an area of small bowel and resulting intestinal fistula. Report on exact nature of first operation not received. Skin at fistula opening excised and held by clamps. Dissection of loops has been completed. Note plications of bowel existing as result of peritonitis. Usable loops of this existing condition not disturbed.

FIG. 8. Same case. Fistula comes from end of proximal end involved in lateral anastomosis. Mesentery had not united at site of anastomosis, leaving a large opening through which a loop of intestine had entered and stuck fast, with developing annular constriction.

FIG. 9. Completion of operation. Fistula has been inverted and is denoted by instrument in upper left corner of photograph. Favorable plications not disturbed but continued with other large loops plicated in such manner that opening in mesentery is covered and subsequent herniation and fixation eliminated.

FIG. 10. A print made from a natural color slide of one of the cases plicated at previous operation from the cecum to the duodenum without interruption. Photographed one year from date of plication at hysterectomy for fibroid. Diet during year has been unrestricted, and weight gain has been 60 pounds, bringing patient up to normal proportions and normal physical activity. This condition of the intestine was not disturbed, and convalescence from hysterectomy was uneventful. There is today, more than a year from the date of the hysterectomy, no abdominal complaint or inability to handle foods.

Complete liberation of all zones of adhesion, evacuation of abscesses, washing off the visceral peritoneum with an antiseptic of the non-corrosive type, plication as is indicated by the condition of the dissected bowel with wing anastomoses around points of annular constriction, through a midline incision, usually without leaving a drain: these constitute the measures taken during this second operation.

In the last ten cases of ruptured appendix complicated with widespread lower abdominal peritonitis, and coming to second operation, eight have survived and have shown no postoperative disability in function or symptom referable to the intestinal tract. Note should be made that not all cases come to second operation since some die forthright and some recover without need of second operation. Some of these latter come in years later with obstruction from a band over the ileum.

We are concerned here solely with those in whom distention persists, in whom partial obstruction remains partial only because of early and continuous activated peristalsis, and in whom mesenteric abscess is expected.

The photographs cannot show the extent of denudation of peritoneum at areas of intestinal constriction, or the mesenteric abscesses, because these are uncovered only by the dissection of the adherent bowel. Color slides showing exudates and finer differentiation of tissues serve much better. However, the chronic distention of the intestine and enough other matter do indicate to the eye the true state of the pathologic process met on opening the abdomen in these cases. The patients used in illustration are well and we feel that they could not be expected to live under any other plan of campaign.

SUMMARY

1. A serious complication of peritonitis from ruptured appendix has been described, photographed, and adequately treated through extension of the use of plication as previously described.

2. Other use of plication has been demonstrated.

3. The value of plication in eliminating certain mortality and morbidity has been shown.

4. The end result of the most extensive plication has been illustrated and described.

CASE OF ENCHONDROMA OF PHALANX SIMULATING GIANT CELL SARCOMA

Saul Alfred Ritter, M.D., F.A.C.S.

Assistant Surgeon, Misericordia Hospital

NEW YORK CITY

SOLITARY cartilaginous tumors, enchondroma and ecchondroma, occur rarely in the phalanges, metacarpal and metatarsal bones. When they do occur they are of considerable clinical importance.

Chondroma is a benign tumor which may arise from cartilage anywhere in the body. Cartilage is embryonal transitory tissue and occasionally undergoes sarcomatous metaplasia. These tumors may be solitary or may arise from multicentric foci. They not infrequently grow from epiphyseal cartilage.

A chondroma when growing outwardly upon the surface of bone is known as ecchondroma; when the tumor grows into the substance of bone it is designated as enchondroma. It may thus cause expansion of the shaft and when combined with cystic degeneration may clinically and roentgenologically resemble giant cell sarcoma (Boyd[1]).

Frankenthal[2] published two cases of enchondroma of the phalanx which were cured by curettement without recurrence. A unique case of chondromatosis of a metacarpophalangeal joint was reported by Linden.[3]

Meyerding[4] of the Mayo Clinic, in 1920, reported eighteen cases of chondromata. They occurred in various sites: nine in the bones of the hand, two in the metatarsal bones, one in the sternum, one in the scapula, one in the lesser trochanter of the femur and three in the knee. The youngest patient was 3 years old, the oldest 53 years. Most of the cases were treated with local currettement; in several cases recurrence was noted, but none became malignant.

Raisch[5] reported a case of a man 56 years old who had a tumor of the proximal phalanx of the right thumb for many years, and suddenly developed metastases in the right radius and soft tissues of the forearm. After resection and eventual amputation of the arm the tumors were found to be chondrosarcoma. Obviously the tumor of the phalanx existed as a benign chondroma for many years before it became malignant and metastasized.

Allende[6] reported a case of chondromata of a metacarpal bone and several phalanges of the left hand of a child of 14 years. It was after several years that the tumor suddenly became malignant, ulcerated and metastasized to the lungs.

CASE REPORT

C. G., female, 70 years of age, complained of pain and progressive enlargement of the proximal phalanx of the fourth finger of the left hand. In 1926, she had accidentally slammed a door upon the left hand. The finger became progressively larger and painful. In 1933, she had received a series of deep Roentgen ray treatments at a local cancer hospital, where the lesion was diagnosed as giant cell sarcoma. She had received this form of therapy at irregular intervals; during June and July, 1936 she sustained in the course of these treatments a severe Roentgen ray burn of the finger which was followed by persistent severe pain.

Upon examination, the proximal phalanx of the fourth finger of the left hand was found to be enlarged to about three times the size of the corresponding finger of the other hand. It was firm, irregular, fusiform in shape and contained a bony tumor which clinically looked like a malignant neoplasm. The finger could not be flexed at the proximal or middle joints. The skin showed a recent diffuse burn on the anterior and posterior aspects of the finger and the margins of the adjacent fingers. No enlarged lymph nodes were present in the antecubital, epitroclear or axillary regions. The pain could not be relieved by narcotics. Roentgen exami-

Ritter—Enchondroma
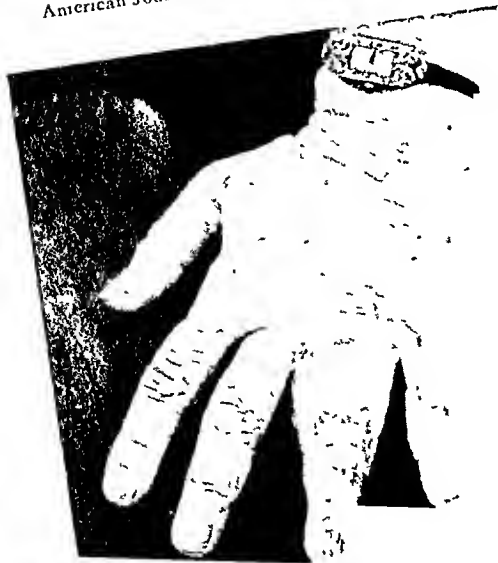
Fig. 1.

Fig. 2.

nation was not considered advisable in the presence of the burn. (Fig. 1.)

On August 10, 1936, I amputated the fourth finger of the left hand through a racket incision. The patient was permitted to go home two days after operation. The sutures were removed after seven days, but it was four weeks before the incision was completely healed. This I believe was due to a lack of vitality of the tissues resulting from the Roentgen therapy.

Observation of the patient for two years subsequently does not reveal any metastases.

Roentgen examination of the finger was made after amputation by Dr. H. B. Phillips. He reported the presence of a cystic bone tumor involving the entire shaft and proximal end of the proximal phalanx. There was fusiform dilatation of the phalanx and considerable subperiosteal proliferation. The cortex of the bone appeared to be perforated on the flexor aspect. The lesion resembled that of a thoroughly irradiated giant cell sarcoma. (Fig. 2.)

Pathologic examination of the amputated finger was made by Dr. Julius Pincus who reported that the entire mass measured 10 cm. in length and 3.5 cm. at the widest diameter. The skin showed brownish discoloration from the tip of the finger to its base. There was a fusiform tumor, apparently well encapsulated, involving the entire phalanx. On cross section, the tumor was soft, cystic and filled with gelatinous material.

Microscopic examination of the bone tumor showed a cartilaginous structure growing inwardly to the medulla of the bone. The bony tissue was necrotic in places and also showed myxomatous degeneration. Newly formed young fiber bone had replaced the normal marrow tissue. No malignant change was noted. (Fig. 3.)

FIG. 3.

The diagnosis was enchondroma of the phalanx.

REFERENCES

1. BOYD, W. Textbook of Pathology, p. 873. Philadelphia, 1932. Lea & Febiger.
2. FRANKENTHAL, L. Arch. f. klin. Chir., 182: 583–589, 1935.
3. LINDEN, O. Acta chir. scandinav., 75: 181–184, 1934.
4. MEYERDING, H. W. J. Orthop. Surg., 2: 77–91, 1920.
5. RAISCH, O. Deutsche Ztschr. f. Chir., 245: 161–164, 1935.

LOW BACK PAIN AND SCIATICA SECONDARY TO A STRAIN IN A PRESPONDYLOLISTHESIS OF THE FOURTH AND FIFTH LUMBAR VERTEBRAE

REPORT OF A CASE

Samuel Kleinberg, M.D., F.A.C.S.

Attending Orthopedic Surgeon, Hospital for Joint Diseases and Lebanon Hospital

NEW YORK CITY

THIS report of a single case is made not because the occurrence is rare or its symptomatology unusual, but to center attention on a lesion the pathology of which is not generally appreciated, and to emphasize the potential disabling effects of a trauma on the soft tissues in a prespondylolisthetic area.

Definitions. It is important at the outset that two terms shall be defined so that we may understand the significance of each of them.

Sciatica. This term implies the presence of pain along the course or in the distribution of the sciatic nerve. There may be an accompanying variable atrophy of the thigh and a diminished or absent ankle reflex. There are no sensory, motor or trophic changes such as are found in a sciatic neuritis. Sciatica, for the present a term of convenience, is the name of a symptom and not of a disease.

Prespondylolisthesis. This term, first used by Dr. Armitage Whitman, has been employed by the present author and others to describe the existence of a congenital laminar defect in one of the lower lumbar vertebrae, it being assumed that the existence of a defect in the posterior arch of one of the lumbar vertebrae predisposes this vertebra to an actual future displacement of its body, or spondylolisthesis. Such an occurrence has actually been described, justifying the use of the term. In November, 1937, at a joint meeting of the New York Academy of Medicine and the Philadelphia Orthopedic Club, Dr. Ferguson of the New York Orthopedic Dispensary and Hospital reported an instance in which a prespondylolisthesis became a spondylolisthesis. He described the case of a girl of 15 years who, at the present time, has a spondylolisthesis, while three years ago, as the roentgenograms definitely show, she had only a laminar defect without vertebral displacement, that is, a prespondylolisthesis. Prespondylolisthesis implies the existence of a bony defect in the posterior arch of the vertebra resulting from lack of fusion of the lamina and pedicle.

When one sprains an interphalangeal joint of a finger it is natural to find pain radiating to the very tip of the finger and also up towards the hand, by reason of pressure on and irritation of the digital nerves. Similarly in other joints a soft tissue lesion, such as a sprain of the external lateral ligament of the ankle or of the medial ligament of the knee, results in pain not only locally but also in structures some distance away from the primary lesion. In exactly the same way an injury to the soft tissues in the lower lumbar area, particularly in the region of the vertebrae, may irritate the fibers entering into the formation of the sciatic nerve and cause sciatic pain in the thigh, leg or foot. The laminar defect in a prespondylolisthesis of the fourth or fifth lumbar vertebra is close enough to the sciatic nerve so that when the connective tissue which bridges the bony hiatus is subjected to an injury the resultant hemorrhage and edema may extend to and irritate the sciatic nerve.

In the case herein reported the neural arches of the fourth and fifth lumbar vertebrae were weakened by the existence

of a laminar defect in each of them. The sudden, violent muscular effort, which under normal conditions, that is, in an

occasions during the previous few years, after strenuous dancing, he had had fleeting pains in his lower back. On August 15, 1937 he was
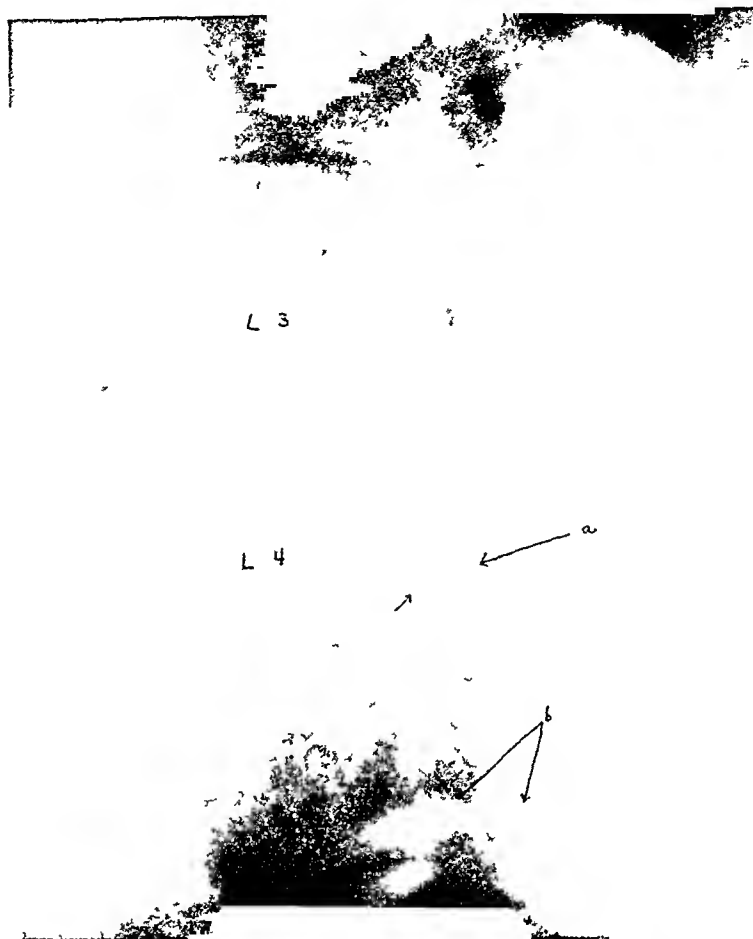
FIG. 1. Prespondylolisthesis of the fourth and fifth lumbar vetebrae. *a* and *b*, osseous laminar defects.

individual with bony neural arches intact, might not have caused any damage, resulted in this patient in a strain of the connective tissues uniting the segments of the neural arch. The pathology of a strain involving not only the laminar bridge of connective tissue but also the nearby soft tissues, consisting of a tear of tissue, hemorrhage and edema, resulted in local pain, or backache, and sciatic pain, or sciatica.

CASE REPORT

A male, 38 years old, a professional dancer, was referred on January 5, 1938 with a chief complaint of pain in the lower part of the back and along both sciatic nerves, aggravated by exercise and relieved by rest. On a number of

engaged in a dancing act in which at one moment he was set with his trunk erect, his lower limbs flexed at the hips and knees, and his arms outstretched, ready to catch on his shoulders two dancing partners, who were to jump down from a slight height. There was a slip-up in the timing and his partners did not jump as they were supposed to. The patient, in a quandary about the situation, arose very suddenly and violently, and as he did so he felt a sharp pain in his lower back. The pain has persisted and has disabled him.

Examination revealed that the antero-posterior motion of his spine was markedly restricted. Forced motion of the spine caused pain in the lower lumbar area and along both sciatic nerves. Straight leg raising on either side was limited and caused low back pain. There was tenderness to pressure over the

lower lumbar vertebrae. The sensory and motor functions and the reflexes were normal. The lateral roentgenogram of the vertebrae showed a laminar defect in the fourth and fifth lumbar segments. (Fig. 1.)

The clinical and x-ray findings led to a diagnosis of a strain of the lower back, weakened by the previous existence of a prespondylolisthesis. The patient was provided with a low back support and was given physiotherapy. He continued at his work of dancing, but omitted the act requiring unusual strain of his back. Within a month he was greatly relieved and discontinued treatment.

CONCLUSION

In this case we have an instance of a congenital osseous defect in the laminae of the lower lumbar vertebrae which established a locus minoris resistentiae. A violent muscular effort strained, that is stretched or tore, the connective tissue bridging the laminar defects, giving rise to backache and bilateral sciatica. Conservative treatment has so far relieved the symptoms in this patient. However, since he has had several attacks of backache, it may be assumed that sooner or later it will become necessary to perform a spine fusion operation to unite the laminae of the third, fourth, and fifth lumbar segments and the first sacral segment and thereby eradicate the weakness resulting from the congenital defect in the posterior arches of the fourth and fifth lumbar vertebrae.

VERY often, pain seems to depend on some excess in respect of quality or duration over the normal action of vaso-constriction or vaso-dilation. It may be brought about, or suppressed, by some abrupt vaso-motor change, whether spontaneous or otherwise.

From—"The Surgery of Pain" by René Leriche (Williams & Wilkins).

SACRO-INGUINAL SINUS

REPORT OF A CASE

JOSEPH W. McHUGH, JR., M.D.

Assistant Surgeon, Lee Hospital

JOHNSTOWN, PENNSYLVANIA

THE following case is noteworthy because of the difficulties which developed after an apparently trivial injury. The incidence of such conditions is believed to be extremely rare and the surgical management a complex problem. The case is one of sacro-inguinal sinus complicated by a simple vertical fracture through the tip of the sacrum and coccyx.

CASE REPORT

A white male, aged 21, a coal miner, was seen following a motorcycle accident in which the handlebar cut his upper right thigh. He complained of severe pain in the lower back and right groin. Examination revealed a deep laceration, undercutting in type, just below and parallel to Poupart's ligament, exposing the femoral vessels. Emergency therapy included débridement and closure with silkworm sutures.

On the day following injury the patient seemed in good general condition. He complained of moderate pain in the right groin, and also of coccygeal pain at times quite severe.

He stated that he had always been in good general health, having had no previous injuries and no operations. He was not alcoholic. There was no history of any familial disease. The temperature was 99, pulse 92, and repiration 18. The patient weighed 165 lb. The blood pressure was 118/78. Other physical findings were normal except for recent injuries. There was pain and tenderness over the coccyx and lower sacral region, but no swelling. There was pain and tenderness over the right groin with moderate swelling. The red cell count was 4,800,000, white cells 8,900, with 76 per cent, polymorphonuclears, 20 per cent lymphocytes, and 4 per cent mononuclears. The urine revealed no pathologic findings.

An x-ray of the sacrococcygeal region revealed a vertical fracture in the midline through the lower sacrum and the coccyx. Position of the fragments was good.

A few days following admission the patient became quite toxic and delirious, with a temperature of 103. Examination of the wound in the right groin revealed increased swelling and sloughing of some of the sutures. The sutures were removed with subsequent gradual improvement of the patient. The wound took on a healthier appearance and began to granulate. Pain in the coccygeal region persisted. Coccygectomy was deemed inadvisable in the presence of infection. Bed rest was the only consideration to the fracture. After several weeks the wound had closed with the exception of a small sinus at the lower angle. This persisted.

Six weeks following injury the patient developed a chill, a rise in temperature, associated with pain in the right lower abdomen. There appeared swelling, tenderness, and fluctuation in the right grain just superior to Poupart's ligament. It was decided to explore this region under anesthesia. Incision and drainage disclosed a profuse discharge of necrotic and purulent material. This resulted in gradual improvement. However, drainage persisted for eight weeks. At the end of that time the wound healed by second intention.

In spite of persistent care the original wound failed to close completely. It was presumed that drainage was inadequate as the patient remained slightly septic, the temperature ranging from normal to 99.3 daily. It was decided to open the primary wound and explore it for pockets. A sinus was found leading into the pelvis, extending beneath Poupart's ligament and over the bladder, rather tortuously. A uterine sound was inserted into the sinus which apparently led to the sacrum. The walls of the tract were curetted and a No. 20 catheter left in situ. The evening following operation was a very stormy one for the patient. He became quite delirious and his temperature

rose to 105. His coccygeal pain was aggravated by our procedure. With general care in a few days there was a decided improvement. The young man, however, had lost 40 pounds in weight since his injury.

Drainage from the wound was very profuse for the first week following the insertion of the catheter. Gradually the discharge diminished and the patient's temperature remained normal. After a month he was discharged to the out-patient department and the wound was irrigated daily with Dakin's solution. This resulted in a very marked general improvement but the sinus persisted.

An x-ray of the tract was taken following injection of bismuth in vaseline. This revealed a tortuous sinus passing from the right groin over the bladder and thence to the mid-anterior surface of the sacrum. The report added that there was a suggestion of bone necrosis in the region of the lower sacrum.

The patient improved as long as the catheter remained in situ. Any attempt to promote healing by withdrawal of it failed. Pain in the lower back persisted. He was advised to submit to an operation for excision of the coccyx. It was thought that bone necrosis might be responsible for infection of the tract.

On October 1, 1935, the entire coccyx and lower portion of the sacrum were excised. They revealed gross evidence of necrosis. The base of the sinus was not explored due to danger of injuring the soft structures. The wound was closed leaving one rubber tube drain in place. Convalescence was uneventful. Coccygodynia disappeared. The patient was discharged from the hospital October 21, 1935 and two weeks later the incision had healed. Drainage from the right groin continued but seemed less profuse. It was irrigated twice weekly with 5 per cent mercurochrome.

In January, 1936, the catheter was gradually withdrawn and irrigations performed once a week. This resulted in improvement. After a few more weeks the catheter was removed and re-inserted periodically for irrigation. Complete closure of the sinus resulted on February 14, 1936. The patient's temperature remained normal, the white blood count was 7,900. He had no complaints except for numbness in the left leg. This was attributed to scar formation in the region of the left iliac vein where there was apparently a mass palpable.

The patient was seen six months following discharge and was in excellent physical condition.

SUMMARY

A case of sacro-inguinal sinus, complicated by fracture of the lower sacrum and coccyx, is presented. Osteomyelitis of the sacrum and coccyx either caused the sinus or prevented its healing. Infection cleared up following removal of the involved bone at the base of the tract. After many months of treatment and observation the patient made a satisfactory recovery with no sequelae.

ASPIRATION OF BLUE DOME CYST*

HARRY C. SALTZSTEIN, M.D.

DETROIT, MICHIGAN

CASE REPORT

AN unmarried nurse of 42, first seen July 6, 1934, had had pulmonary tuberculosis fifteen years before, with apparently satisfactory arrest after three months bed rest at home. Her menses had been regular every twenty-six days, with no pain, the flow lasting from two to four days. She complained of migraine headaches, and there had been slight serous discharge from the nipples a few days before each period for several years.

Her present complaint was a lump in the left breast which had developed suddenly four days before. There had been no previous discomfort, no previous trouble with the breast, and no swelling during the menses.

There was a firm irregular dense mass ¾ inch in diameter at the outer tail of the left breast, 2½ inches from the nipple. It lay superficially underneath the skin, moved easily, and was not attached to the skin, but was somewhat attached to the breast tissue about it.

A radial incision was made in the outer portion of the left breast, widely excising the tumor mass (July 9, 1934). The cut surface showed a typical blue dome cyst. Two or three cysts were entered, discharging thin fluid.

Microscopic examination showed numerous small cysts, apparently well encapsulated, scattered through an indurated mass of breast tissue. The microscopic diagnosis was chronic cystic mastitis.

The patient was well until January, 1935. Then she noted another cyst in the right breast. This was aspirated one week after onset, and 10 c.c. of amber colored fluid was obtained. The breast tissue was thereafter normal in texture.

Six months later, and one year after the original operation on the left breast, the patient noticed an indurated mass in the left breast below the previous upper outer quadrant scar. This was excised under local anesthesia in the office (July 11, 1935). Microscopic examination showed chronic cystic mastitis.

She was again well for a few months. Then (October, 1935) she noticed a small swelling in the outer quadrant of the right breast where aspiration was done one year before. A small firm thickening was noted at this site. This was watched for six months, and as the mass seemed to grow a little larger and felt cystic at one spot, it was excised (April 15, 1936). A large irregular mass was removed including all of the upper outer quadrant of the right breast tissue. The mass was cystic. One blue dome cyst was punctured, evacuating clear fluid. The microscopic diagnosis was chronic cystic mastitis.

Trouble recurred in the left breast two months later (June, 1936), when a cyst came up suddenly in the mid-portion of the left breast, toward the center from the previous scar, and apparently entirely distinct from the old involved area which had been excised. On July 25, 1936 it was aspirated and 20 c.c. of thick glairy greyish opaque material was removed.

There has been no reaccumulation and the patient has been well since. When last seen (February 1, 1938) menses were still regular, with moderate flow for four days and no pain.

The right breast was smooth, soft, and supple, but there was a heavy scar in the outer upper quadrant. The left breast showed a soft broad scar in the upper outer quadrant and some induration between the scar and the nipple, though this seemed less than before. The upper midportion, where the cyst was aspirated, was soft and smooth.

SUMMARY

The history of a patient in her early forties is recorded. She had had no previous breast abnormality except slight serous discharge before menses in earlier years which had subsided. A blue dome cyst suddenly appeared in the left breast and local excision was done in 1934. A second local excision of an adjoining portion of the left breast was done one year

later (1935). Another cyst was aspirated in 1936. Two years later she was well. In the right breast, a cyst was aspirated in January, 1935.
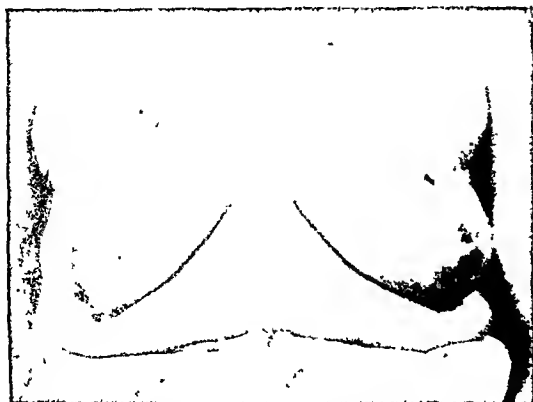
FIG. 1. Chronic cystic mastitis. Multiple local excisions and aspirations. Two local excisions in lateral portion of left breast. Aspiration of another cyst at black mark in mid-portion of left breast. Well two years. Aspiration of another cyst in right breast. Local excision later because of persistent induration.

Induration persisted in the outer upper quadrant of the right breast. In January, 1936 excision of a wide area in upper outer quadrant was done. The patient has remained well since.

DISCUSSION

Frank S. Mathews, in a paper read before the New York Surgical Society in 1935, reported that he had aspirated fifty cysts of the breast in twenty years, and added that: "There seems a very strong prejudice on the part of surgeons against aspirating cysts. One is usually told that it is dangerous teaching. None of the surgeons with whom I have discussed the subject admit that they ever resort to aspiration. No paper which I have seen which considers the subject of treatment mentions aspiration of cysts, even to condemn it."

Breast cysts constituted 8 per cent of 664 breast cases reviewed by Adair. They appear usually in the late thirties or early forties, and tend to become smaller as the mammary tissue atrophies after the menopause. They may appear with one period and completely disappear before the next menstrual phase. A relationship between cysts of the ovary and breast has often been noted.

If the contents are bloody, all authorities agree that operation should be done, since the danger of cancer is real—48 per cent in Adair's series.

If there is muddy colored fluid, it is not always cured by aspiration, and infected straw-colored fluid always refills.

If, however, the contents are clear straw-colored or opalescent, it is a perfectly benign condition with no danger of associated malignancy. Adair cured 85 per cent by simple aspiration. Others have concurred in this opinion or reported series of cases followed up and no future malignancy observed (Mathews, Pickhart, Klingenstein, Bull, Abbe, Cheadle and Cutler) so that aspiration of a simple cyst containing amber or opalescent fluid is quite safe and rational.

The breast tissue should be flat and no induration should be left after aspiration. If the cyst refills promptly, it is best to resort to surgery (Mathews).

CONCLUSION

A case of chronic cystic mastitis is reported. Multiple local excisions were done; also cysts in each breast were aspirated. Indications for aspiration are discussed.

REFERENCES

MATHEWS, F. S. Aspiration of breast cysts. *Ann. Surg.*, 104: 220–226 (Aug.) 1936.
CHEATLE, G. L., and CUTLER, MAX. Tumors of the Breast. Philadelphia, 1931. Lippincott.
CRILE, G. W. An analysis of 1347 cases of malignant tumors of the breast with special reference to management and end results. *Cleveland Clin. Quart.*, 1: 49–57 (April) 1932.
PICKHARDT, O. C. Chronic cystic mastitis. *Ann. Surg.*, 96: 151–153, 1932.
KLINGENSTEIN, P. Cystic disease of the breast. *Ann. Surg.*, 101: 1144–1152 (May) 1935.
ADAIR, F. E. Etiological factors of mammary cancer in 200 women. *New York State J. Med.*, 34: 61–68 (Jan.) 1934.
ADAIR, F. E. Diagnosis and treatment of breast lesions. *New England J. Med.*, 212: 336–340 (Feb. 21) 1935.
ADAIR, F. E. Discussion of Mathews paper above.

SARCOMA OF THE VULVA*

Phineas Bernstein, m.d.

NEW YORK CITY

THE high mortality and the certain fatal outcome represented by a vulvar sarcoma is well known. The case here described is unusual in that the patient is alive and well four years after the tumor was removed. The absence of metastasis over this long period as well as the rarity of this malignancy warrant the presentation of facts which follow:

S. K., an obese, 45 year old Russian born housewife, was admitted to the gynecologic ward, January 2, 1934. She had had five normal pregnancies. Menses were always normal before the onset of the menopause one year prior to admission. For sixteen years the patient had observed the slow growth of a nodule in the left labium, until it reached the size of a large grapefruit. There had been periods of two to five years during which no enlargement occurred; in the intervals, however, the size increased and the mass became inflamed and ulcerated. Recently she had experienced difficulty in walking because of the tumor. There were no other complaints or symptoms.

Examination. A firm, nodular, grapefruit-sized mass was found broadly attached to the left vulva. (Fig. 1.) The clitoris at the upper margin of attachment was concealed behind the mass. The flattened left labial fold surmounted the anteromedial surface. The overlying skin was tense, movable, and ulcerated on the inner aspect. The inguinal glands were not enlarged. The general physical findings were normal. Wassermann and Kahn tests were negative. Blood pressure was 128/78, hemoglobin 84 per cent, sedimentation rate seventy minutes. A diagnosis of fibrolipoma was made.

Operation and Findings. Under gas, oxygen and ether anesthesia a longitudinal incision through the skin was made 2 inches lateral to the labial edge. The tumor was removed by both blunt and sharp dissection. Its fossa completely covered the descending ramus of the pubis, reaching the vaginal wall medially. The tumor and its capsule were removed completely after several large vessels were ligated, and the skin was then closed. (Operation was done by Dr. U. J. Salmon.)

Pathologic Report. The specimen was described as grapefruit-sized (10 × 8 cm.), moderately edematous, soft and pliable, and gray-white in color. The capsule was thick and injected and contained several areas of hemorrhage. Large gaping blood vessels were noted.

When sectioned, the cut surface appeared glossy, edematous and white. It was smooth throughout except for several small loculi and a discrete, round yellow area, containing tiny, coalescent, irregular elevations, punctate in character.

Microscopic sections were reported by Dr. Paul Klemperer as polymorphous cell scarcoma of the vulva.

Postoperatively, x-ray radiation, totalling 1200 R units was given directly over the left vulva. It is now four years since operation and no complaints referable to the original condition have been reported by the patient. The chest, pelvic bones and lower spine, recently x-rayed, revealed no metastasis.

DISCUSSION

The literature contains about 100 reports of cases of malignancies of this category. The mortality is very high.

Fraenkel[10] states that the incidence of 0.01 per cent, inclusive of melanosarcoma. E. Kehrer, in the Veit-Stoeckel Handbuch,[13] collected from the literature up to 1929 seventy-seven vulvar sarcomata, excluding melanosarcoma. In 62 per cent they arose from the labium majus, in 11.4 per cent from the labia minora. Sarcomata have also been found in the round ligament,[19] in fibromata,[15,16] in Bartholin glands,[2,12] and in vulvar elephantiasis.[5] Taussig[31] believes that many are derived from the fibrofatty substance of the vulva rather than the fibrous element alone.

Histologically, the type described are spindle,[11] round,[32,33] giant,[29] and polymorphous cell.[19] Myxosarcoma,[23,31] fibro,[4] lipofibro,[14,30] and lymphosarcoma,[31] are reported. Endothelial,[6] perithelial,[25,26,3] and angiomatous varieties are mentioned.

Blair Bell,[1] in twenty-one cases, found the myxosarcoma most common. In a group of twenty-six collected cases, R. T. Frank[8] states there were ten myxosarcomata, four fibro-, six spindle-, three polymorphous, and one each of peri, endothelial, and angiosarcoma.

Taussig[31] feels that many of the round cell sarcomata would now be called lymphosarcomata, in view of the recent changes in the malignant tumor classification.

The age incidence is agreed to be between 30 and 50 years. The average age of incidence is 40 years. However, H. S. Morgan[20] reports one case in a child of sixteen years.

R. T. Frank[8] states that these tumors resemble fibromyomata until ulceration and infiltration takes place. They begin as small pedunculated or sessile growth, and may spread locally in the early stages. They differ from carcinoma and melanosarcoma in that the lymphatic vessels are rarely involved until very late.
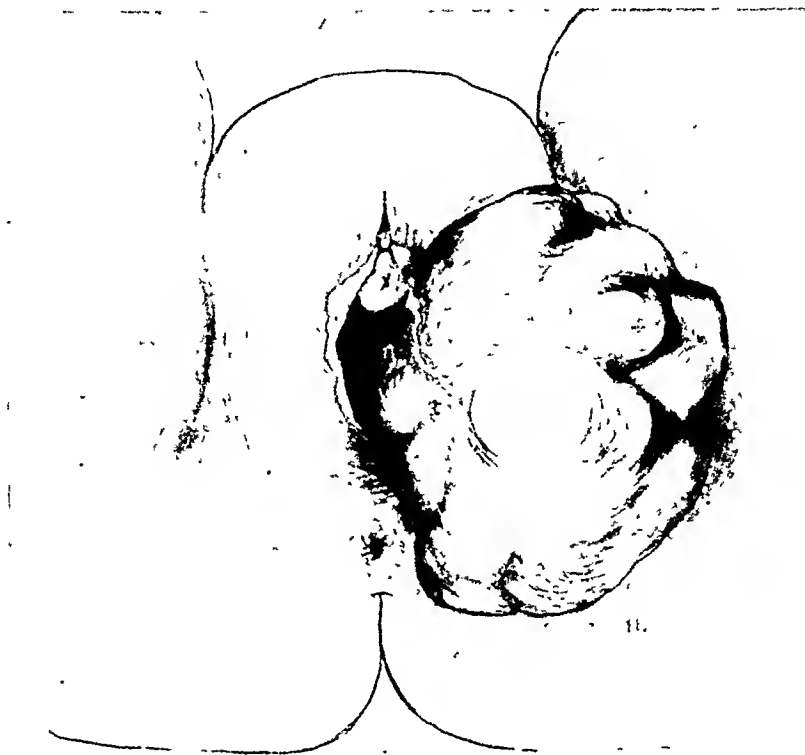
FIG. 1. Sarcoma of left vulva.

Local metastases are found both early and late. Nebesky,[21] found extension in four cases of urethral sarcoma, secondarily to the vulva, vagina and inguinal glands. B. P. Watson's case,[31] was similar. Distant metastases are the rule, however, especially after operation and also late in the disease.

Maas and Olshausen[15] have reported a case with five year freedom from recurrence and Rhomberg[24] an even longer period.

The prognosis is always grave, since the disease is usually fatal because of metastases to vital organs. As regards recurrence after excision, the prognosis is also bad. In five instances, E. Kehrer[13] reported that gestation exercised no influence upon the growth of this tumor.

Diagnosis is rarely made clinically. The microscopic picture is always necessary and final.

Treatment. Early excision offers the only hope of cure. Recurrent local lesions should be repeatedly excised. The value of radium and x-ray seems quite questionable. Surgical procedures employed are usually simple and not attended by serious sequelae.

SUMMARY

1. Sarcoma of the vulva is a rare disease: about 100 cases are reported in the entire literature.

2. The mortality is exceedingly high.

3. A brief résumé of the characteristics of this malignancy is given.

4. The case presented is unusual in that four years after operation, there are (1) no complaints referable to the original tumor; (2) x-rays reveal no metastasis, (3) there are no nodes palpable externally. The patient is clinically cured.

5. A survey of the limited literature and a brief discussion of the clinical course of the disease is also given.

REFERENCES

1. Bell, B. *J. Obst. & Gynaec. Brit. Emp.*, 12: 275, 1907.
2. Bluhm. *Arch. f. Gynaek.*, 71: 1, 1904.
3. Carbone. Un caso di sarcoma della vulva a tipo periteliale. *Arch. obstetr.*, 13: 84, 1926.
4. Caruso. *Soc. ital. di ostet. e ginec.*, vol. 2, 1896. *Zentralbl. f. Gynäk.*, 20: 908, 1896.
5. Chrobak. Tumor der Klitoris (Elephantiasis). *Wien. klin. Wchnschr.*, 1895.
6. Delle Chiage. *Ann. d. ostet. e ginec.*, 29: 451, 1907.
7. Flatau. *Zentralbl. f. Gynäk.*, 27: 990, 1903.
8. Frank, Robert. Gynecological and Obstetrical Pathology. New York, 1922. D. Appleton-Century Co., p. 125.
9. Francke. *Virchow's Arch.*, 154: 363. (First case.)
10. Fraenkel. *Zentralbl. f. Gynäk.*, 28: 1025, 1904.
11. Hartmann. *Zentralbl. f. Gynäk.*, vol. 32, no. 2, 1908.
12. Hofmeier. Schroeder's Handbuch d. Frauenkrankheiten. 14th ed., 1908, p. 65.
13. Kehrer, E. In Veit-Stoeckel. Handbuch der Gynäkologie 5, Part I, pp. 496–503. (Literature.)
14. Kleeberg. *Petersburg med. Ztschr.*, 15: Part 2, 1869.
15. Lichtenstein. *Gynäk. Ges.*, Leipzig, 1908.
16. Lichtenstein. Beitrag zur Frage der Kontagiosität des Condyloma acuminata. *München. med. Wchnschr.*, p. 270, 1922.
17. Lynck, Frank. Pelvic Neoplasms. New York, 1924. D. Appleton-Century Co., pp. 36–41.
18. Maas. Ueber d. Malignität d. Carcinoma u. Sarkome an der ausseren weiblichen Genitalien. Inaug-Diss., Halle, 1887.
19. Maly. *Arch. f. Gynäk.*, 76: 175, 1905.
20. Morgan, H. S. *Am. J. Obst. & Gynec.*, 15: 861–865 (June) 1928.
21. Nebesky. *Arch. f. Gynäk.*, 93: 539, 1911.
22. Netzer. *Zentralbl.*, 41: 413, 1925. (Literature on sarcoma vulvae.)
23. Robb, H. Myxosarcoma of clitoris. *Johns Hopkins Hosp. Rep.*, 2: 231, 1900.
24. Rhomberg. *Zentralbl. f. Gynäk.*, 39: 780, 1915.
25. Schmidlechner. Carcinoma clitoris. *Arch. f. Gynäk.*, 74: 195, 1904.
26. Schmidlechner. *Arch. f. Gynäk.*, 74: 200, 1904.
27. Schmidt, H. R. Spindelzellensarkom der weiblichen Urethra. *Zentralbl. f. Gynaek.*, 50: 3122, 1926.
28. Shaw, A. F. B. *J. Path. & Bact.*, 43: 277, 1936.
29. Szili. *Beitr. z. klin. Chir.*, 31: 734.
30. Taussig, F. J. Diseases of the Vulva. New York, 1930. D. Appleton-Century Co., p. 176.
31. Taussig, B. P. *Am. J. Obst. & Gynec.*, 33: 1017 (June) 1937.
32. Watson, B. F. *Am. J. Obst.*, 69: 4, 1914. (Literature has 52 undoubted cases.)
33. von Winckel. Pathologie der weiblichen Sexualorgane. Leipzig, 1878.
34. von Winckel. Pathologie der weiblichen Sexualorgane. 1881, p. 275. (Collected 54 cases.)

SO-CALLED "LIVER DEATH" SYNDROME FOLLOWING THORACOSTOMY

JOHN GEORGE SLEVIN, M.D.

Assistant Surgeon, Grace Hospital

DETROIT, MICHIGAN

IT was in 1924 that Charles Gordon Heyd[8] first announced the syndrome which has since been termed "liver death." As Sutton[14] describes it, "the so-called high temperature, liver death syndrome is a clinical entity characterized by rapid progressive development of high fever, fall in blood pressure, circulatory collapse, coma and death, with temperature rising as high as 109°F. within thirty-six to forty-eight hours after operation."

So-called "liver deaths" with few reported exceptions have occurred after surgery on the biliary tract. Although it is only thirteen years since the syndrome of "liver death" was first recognized as a clinical entity, there have accumulated in the literature more than 100 cases of this kind. A search of the literature does not reveal a single case of high temperature death following thoracic surgery.

The only demonstrable pathology in "liver deaths," as determined at post-mortem examination, is passive congestion, softening and diffuse disorganization of the liver cords with widespread focal necrosis of the liver. Experimental work done on dogs by Sutton[14] showed that similar clinical causes and living changes were produced in six of fourteen dogs by the ligation of the hepatic arteries. In eight dogs which did not die, adequate collateral circulation to all lobes of the liver was found and there was no hepatic necrosis. Crile[5] believes that there is also interference with the heat regulating mechanism of the body. However, this has not been proved.

The following case fits the definition of "high temperature, liver death syndrome" in that thirty-one hours after operation the temperature began to rise rapidly, there was circulatory collapse, followed by coma and death occurred just thirty-five and three-quarter hours after operation.

CASE REPORT

J. B., male, aged 60 years, white, Polish, a baker by trade, contracted dry pleurisy of the lower right pleura on November 9, 1936, and was treated symptomatically. Three weeks later (December 3), he developed a right lobar pneumonia. He was treated at home and was making a normal recovery until December 15, when his temperature began to rise and he complained of pain in the right chest at the base. A diagnosis was made of delayed resolution of the pneumonia associated with an encapsulated empyema of the right pleura.

The patient was admitted to the Grace Hospital, Detroit, on December 22, 1936. X-rays taken on admission confirmed the diagnosis of pleural empyema and unresolved pneumonia.

On admission the chest showed some swelling of the lower right wall with obliteration of interspaces. The lungs exhibited diminished excursion on the right side and absent fremitus at the right base. Flatness on percussion was noted from the eighth right rib to the base with absent breath sounds in the same area. Above the eighth rib right posteriorly, breathing was vesicular. Bronchovesicular breathing and many crepitant râles were heard over the left lower lobe. The heart was negative except for a slight shift to the left.

Hemoglobin was 15 Gm. (90 per cent); erythrocytes numbered 4,380,000; leucocytes, 11,400, with polymorphonuclear cells 81 per cent, (filaments 56 per cent, non-filaments 25 per cent), small lymphocytes 15 per cent, monocytes 1 per cent, and metamyelocytes 3 per cent. No abnormal red blood cells were seen. Sputum showed no pneumococci present. Culture of pus from thoracentesis was positive for

pneumococci. Urinalyses on December 22 and 24 were essentially negative.

On December 24, under novocaine infiltration anesthesia, thoracostomy was done. The empyema cavity was opened and two soft rubber drains inserted. About 400 c.c. of thick pus was drained off at the time of operation.

For the next twenty-hours the patient's condition was satisfactory. The following morning, however, the patient was cyanotic and required oxygen therapy. His pulse was 128 and was weak. He was mildly delirious. He was catheterized at 1:30 P.M. and 300 c.c. of urine was obtained. This was all the urine passed in sixteen and one-half hours and the patient did not void thereafter. Coma developed about 3 P.M., and shortly thereafter pulmonary edema appeared. The patient's temperature rose rapidly. At 8 A.M. it was 102°F.; at noon 102.8°F.; at 4 P.M. it was 107; and just before death it was 108. At noon the pulse was 150 per minute. Digalen was given without effect. Thereafter, the pulse was too weak to count. The patient expired in coma at 8:45 P.M.

DISCUSSION

Heyd,[11] in his most recently published article on this subject, gives a revised classification of liver deaths, viz:

"1. Those associated with hyperpyrexia and coma—a rapidly developing lethargy, stupor and coma, death terminates the picture in from eighteen to thirty-six hours.

"2. Liver deaths in the presence of a constantly diminishing jaundice—slowly developing stupor and coma; final clinical picture is similar to 'cholemia death' from cirrhosis of the liver.

"3. Liver deaths associated with some unrelated kidney disease—anuria is a factor in the terminal picture. Forty-eight hours after operation the clinical picture is not dissimilar to shock, with cold, clammy skin; failure in water elimination and a marked rise in non-protein nitrogen."

Thus, according to Heyd's classification the case herein reported readily falls into group 1.

As has been said, most of the cases of "liver deaths" follow biliary surgery. But in 1935 Connell[4] reported five instances of high temperature deaths occurring after operations other than on the biliary tract. And in 1937 De Courcy[6] reported two cases of "liver death," one following colostomy and the other after hysterectomy for simple fibroid.

Boyce and McFetridge[1] recently reviewed a number of casually selected autopsies covering burns, intestinal obstruction and thyroid disease and were struck with the resemblance of these cases to the hyperpyrexic and hepatorenal deaths following biliary surgery.

The fact that "high temperature, liver deaths" can occur following surgery other than on the biliary tract, brings up for consideration the factor or factors responsible for this fatal syndrome.

LIVER DEATHS OF TOXIC ORIGIN

The prevailing opinion regarding the cause of "liver death" is that it is of toxic origin. Boyce and McFetridge,[2] studied thirty-four cases of this type and performed experiments on animals, which included the injection into dogs of saline and aqueous extracts made from the liver of patients who died with the typical "liver death" syndrome. As a result they came to the following conclusions:

"1. The underlying factor is some degree of hepatic damage, either preëxistent or the result of direct trauma;

"2. When the added strain of operation is superimposed on the existing hepatic disability, the damaged liver cells release into the circulation some potent, water soluble toxic substance."

If we accept this theory as a working hypothesis, is it the final explanation of the underlying factor of liver death? It is possible that another factor enters into the picture, a factor, perhaps, which exists only in vivo, not discernible at autopsy.

What caused the "liver death" in the case herein presented? As far as could be determined clinically, there being no autopsy, there was a minimum of liver damage such as might be expected following a protracted pneumonia and concomitant empyema. The operative procedure

was simple and should have lessened the toxemia. Nevertheless the patient died a typical "high temperature, liver death."

On the basis of Sutton's[17] experimental work on the ligation of the hepatic artery with the production of a typical liver death syndrome, a thrombosis or embolism of the hepatic artery could be the cause of death in this case.

However, thrombosis of the hepatic artery is so rare, there being only two authentic cases on record, that the results of this sort of accident are unknown. Embolism of the hepatic artery is also comparatively uncommon. Either thrombosis or embolism of this artery would produce approximately the same pathology as was experimentally produced by ligation, i.e., necrosis of the liver.

Thrombosis of the hepatic veins is also rare and would not be likely to produce the same effect as we observe in "liver death."

While thrombosis or embolism of the hepatic artery is a possibility, it is a rather remote likelihood.

There is another way, however, by which the hepatic circulation, theoretically at least, could be occluded, namely, through the sympathetic nervous system.

AUTONOMIC SYSTEM'S RÔLE IN LIVER DEATHS

Mann,[13] in a recent review of the physiology of the liver, remarks that the sympathetic nerves are present in the liver in great abundance; that they reach the liver by accompanying the hepatic artery and seem to be mainly for the purpose of controlling the blood flow through this artery.

The liver receives its sympathetic nerve supply from the celiac ganglion. This ganglion, in turn, receives its branches from the sixth to the twelfth thoracic segments of the spinal cord. The intercostal nerves involved at the thoracostomy operation arise from the sixth and seventh thoracic segments. Furthermore, the fibers of the intercostal nerves and of the sympathetic system emerge together in the

anterior nerve roots, the sympathetic leaving the spinals later by way of the white rami communicantes. Thus it is evident that these two systems are closely related.

Mann further states "the arterial blood flow to the liver is under vasomotor control and is essential for the existence of the organ." Hence, stimuli received by the intercostal nerves could be relayed to the autonomic nerves innervating the hepatic artery, by way of the sympathetic fibers which take origin from the same cord segments.

If this happened the hepatic artery could undergo a massive and prolonged spasm as a result of "referred stimuli," producing a decided diminution in blood supply to the liver. It will be recalled that this patient's liver was already damaged by prolonged infection and the diminution in blood supply could easily institute necrosis of the liver.

Thus the conclusions which Boyce and McFetridge[2] arrived at, are fulfilled in this case—a damaged liver with the added strain of operation producing a toxin which is liberated into the blood stream.

The hypothesis here presented is offered as an attempt to fix the mechanism whereby "the strain of operation" affects the "damaged liver cells."

This is pure theory, unsupported by any clinical or experimental evidence. But if true, it would solve the problem of what causes "liver deaths."

CONCLUSION

This, and similar cases of extrahepatic "liver deaths" should crystallize our attention on the fact that "liver deaths" do occur after operations on other parts of the anatomy than the biliary tract; that in preparing patients for operation we should consider the possibilities of liver damage and give the patient a chance to build up a sufficient store of liver glycogen to withstand the rigors of operation. Also, where it is known that some degree of liver damage already exists, special care should be taken

to prevent "liver death" by conserving the glycogen reserve of the liver, especially by a more liberal use of intravenous glucose solution postoperatively.

REFERENCES

1. Boyce, F. F., and McFetridge, E. M. "Liver deaths" in surgery; analysis of 34 cases with new explanation of clinical and pathological picture. *New Orleans M. & S. J.*, 88: 563–567 (March) 1936.
2. Boyce, F. F., and McFetridge, E. M. "Liver deaths," experimental study of changes in the biliary ducts following decompression of obstructed biliary tree. *Arch. Surg.*, 32: 1080–1086 (June) 1936.
3. Boyce, F. F. So-called liver death syndrome in surgery. *Surg., Gynec. & Obst.*, 61: 122, 1935.
4. Connell, F. G. Liver deaths, rapid high temperature deaths. *Ann. Surg.*, 100: 319, 1934.
5. Crile, G. W. Critical facts in surgery of the gallbladder and ducts. *South. M. J.*, 20: 85, 1927.
6. DeCourcy, J. L. "Liver deaths" in general surgery. *Ann. Surg.*, 106: 58–61 (July) 1937.
7. Eiss, S. Conservation of hepatic function in gallbladder operation; precautionary measures to prevent "liver deaths." *Ann. Surg.*, 98: 348–353 (Sept.) 1933.
8. Heyd, C. G. Liver and its relation to chronic abdominal infections. *Ann. Surg.*, 79: 55, 1924.
9. *Idem.* Significance of liver function from surgeon's standpoint. *New York State J. Med.*, 33: 1317–1320, 1933.
10. *Idem.* "Liver deaths" in surgery of the gall-bladder. *J. A. M. A.*, 97: 1847–1848 (Dec. 19) 1931.
11. *Idem.* Liver deaths and the complications of gallbladder surgery. *South. Surg.*, 6: 3 (June) 1937.
12. Helwig, F. C. Recent advances in the knowledge of the pathology of the liver. *Am. J. Surg.*, 19: 462 (March) 1933.
13. Mann, F. C. Review of experimental investigation from surgical viewpoint. *Minnesota Med.*, 19: 695–702 (Nov.) 1936.
14. Sutton, Jr., J. E. Acute postoperative necrosis (so-called high temperature, liver death syndrome). *Am. J. M. Sc.*, 192: 219–224 (Aug.) 1936.
15. *Idem.* "High temperature, liver death" syndrome. *Proc. Soc. Exper. Biol. & Med.*, 32: 712, 1935.
16. Schutz, C. B., Helwig, F. C., and Huhn, H. P. A contribution to study of so-called liver deaths. *J. A. M. A.*, 99: 633, 1932.
17. Walters, W. Function of liver in relation to surgery. *Ann. Surg.*, 94: 15, 1931.

RUPTURE OF THE SPLEEN FROM MUSCULAR ACTION

REPORT OF A CASE

L. M. RANKIN, M.D., F.A.C.S.

PHILADELPHIA, PENNSYLVANIA

IT is interesting to observe that the first recorded case of splenectomy, according to Krumbhaar[1] was by Zacearelli of Naples in the year 1549. It is, also, of more than passing interest to know that the first case of splenectomy reported in this country was for injury due to a knife wound and was reported by O'Brien in 1816.[1] Since then there have been advanced and reported many causes of rupture of the spleen from spontaneous rupture of a normal spleen to that resulting from a very severe trauma or injury. While there has been more or less of a controversy as to the possibility of a normal spleen rupturing spontaneously, it has been reported by some writers and is now recognized as having occurred. Zuekerman and Jacobi,[2] in May, 1937, made an exhaustive critical study of the medical and surgical literature and found only twenty genuine recorded cases of spontaneous rupture of the spleen and seven questionable cases.

Bailey,[3] in 1930, reporting one case of spontaneous rupture of a normal spleen said, "Only the spleen can behave in this curious manner and from the practical standpoint there follows a good aphorism, 'In atraumatic hemoperitoneum in the male examine first the spleen.'"

One is impressed with the large variety of trauma that has produced a rupture of the spleen. The causes appearing most frequently were falls, pressure or compression, kicks or blows over the spleen, crushing injury, football accidents, and accidents from sledding and from "belly-flopping." In this modern age of automobiles and speed, the automobile is producing more ruptured spleens than any other one cause.

Muscular contraction with increased intra-abdominal pressure, fortunately, is a very rare and little mentioned cause of traumatic rupture of the spleen but one that must not be lost sight of. Susman,[4] in 1927, reported one case in which a man suddenly developed severe pain in the upper abdomen when he bent down to pick up a bucket of water. There was no history of any injury or blow prior to this date, although there was a history of flatulence and indigestion for the previous three months. Operation revealed a ruptured spleen and the patient died three days later. In 1933 Bohler[5] reported a case in which he believed muscular contraction and increased intra-abdominal pressure were responsible for the rupture of the spleen. A patient, in health, suddenly developed upper abdominal pain when bending over to pick up a heavy object. Operation revealed a ruptured spleen.

CASE REPORT

The patient had been sent to the hospital by his family physician with a diagnosis of acute appendicitis, but the internes on service thought he had a perforated gastric or duodenal ulcer.

The chief complaints were pain in the left upper quadrant and weakness. The family history was more or less negative. Father and mother had died of peritonitis. Three brothers and three sisters were living and well. The patient declared that he had never been sick, had never been operated on, and had never been injured.

About 8:30 A.M., while he was at work, he strained to pull a cable over a pulley in order to move or raise a heavy object and suddenly experienced severe pain in the upper left quadrant. He felt very weak and dizzy and had the sensation of fainting. He was cold, sweated profusely and his weakness and prostration increased. He was taken home and his family physician called. The latter diagnosed acute appendicitis and late in the afternoon sent the

patient to the hospital for operation. By this time the man had pain in his left shoulder.

The abdomen was tender and very rigid, but there was not a definite "boardlike" rigidity. Urinalysis was negative; the white count was 13,300 with 83 per cent polymorphonuclears. My first diagnosis, which later proved to be correct, was a ruptured spleen, but we were dealing with a very intelligent person who denied any injury whatsoever, and we therefore ruled this out and considered a presumptive diagnosis of perforated ulcer.

The man was operated on immediately, by way of an upper right rectus incision. At the opening of the peritoneum blood ran out. The incision was enlarged and the ruptured spleen palpated. It was necessary then to close the wound and remove the spleen through a left rectus incision.

Following operation the patient was given 300 c.c. of citrated blood. Except for an attack of bronchitis, recovery was uneventful. The patient was discharged on October 2, 1937. He was seen in the follow-up clinic on January 26, 1938 at which time he was symptom-free. His red blood count was 4,680,000, leucocytes 9,200, and platelets 612,000.

The pathologist's report of the removed spleen may explain the predisposing factor to the rupture of the spleen. The report gave the weight as 240 Gm. and showed, in addition to the traumatic rupture of the spleen and capsule, early chronic congestion. V. H. Moon[6] in a study of the variations of the size of the spleen from 2,000 specimens removed at autopsy stated that he did not consider a spleen pathologic that weighed less than 350 Gm., or rather he considered all spleens weighing 350 Gm. or over to be pathologic. Therefore, one would hesitate to call this specimen grossly pathologic.

The congestion demonstrated histologically must have been a predisposing factor. Susman[4] states that Wohl points out that histologically the spleen ages early and believes that these early degenerative changes, softening of the spleen and congestion, are three causes that predispose to its rupture.

CONCLUSIONS

1. Muscular action plus increased intra-abdominal pressure may cause a spleen to rupture. One case is herewith reported.

2. This should be kept in mind to prevent making an error in diagnosis where the symptoms are suggestive of a ruptured spleen but the history is negative for injury.

3. When one encounters blood in the peritoneal cavity in the male, in the absence of trauma, examine first the spleen.

4. Kehr's sign, pain referred to the left shoulder, was present in this case.

5. Chronic congestion is, perhaps, a predisposing factor to splenic rupture.

REFERENCES

1. KRUMBHAAR, E. B. History of the extirpation of the spleen. *New York State J. Med.*, Feb., 1915.
2. ZUCKERMAN, C., and JACOBI, M. Spontaneous rupture of the normal spleen. *Arch. Surg.*, 34: 917–928 (May) 1937.
3. BAILEY, H. Spontaneous rupture of the normal spleen. *Brit. J. Surg.*, 17: 417–421 (Jan.) 1930.
4. SUSMAN, M. P. Spontaneous rupture of the spleen. *Brit. J. Surg.*, 15: 47–54 (July) 1927.
5. BOHLER, E. Eclatement du hile de la rate au 7e mois de la grossesse. *Bull. Soc. d'obst. et de gynêc.*, 22: 707 (Oct.) 1933.
6. MOON, V. H. Racial variation in size of spleen. *Arch. Path.*, 5: 1040–1043 (June) 1928.

AMBULATORY TREATMENT OF FRACTURES OF METATARSALS IN BOTH FEET BY THE USE OF SPONGE RUBBER PADS

Nathan H. Rachlin, m.d.

Attending Orthopedic Surgeon, Beth Moses Hospital

BROOKLYN, NEW YORK

IN fractures of the lower extremity, involving one limb only, patients can be made ambulatory with the aid of crutches, thus eliminating weight bearing on the injured limb. However, when both limbs are injured, getting about becomes either impossible or extremely painful, with the added danger of subsequent malunion or nonunion of the fragments. Enforced recumbency is not the most desirable method of treatment, nor is it the most satisfactory to the patient.

The writer was confronted with this problem recently, and has tried to solve it in the manner described herewith.

CASE REPORT

C. B., age 31, was seen December 22, 1937. Two days previous he had fallen from a step ladder and landed on the plantar surfaces of both feet.

Both feet were swollen and tender, on the plantar and dorsal aspects; weight bearing of the slightest degree caused excruciating pain.

X-rays taken the day of the accident showed a transverse fissure fracture through the neck of the middle metatarsal of the right foot (subperiosteal type) with no displacement or deformity. There was also a comminuted fracture of the head and neck of the second metatarsal with slight inward and dorsal tilting of the distal small fragments.

There were also transverse subperiostea fractures of the necks of the second and third metatarsals of the left foot, but with no displacement or deformity.

Recumbency and elevation of the limbs were advised to reduce the swelling. The feet were padded with sheet cotton and bandaged.

A week later the patient was feeling better, swelling was reduced, but weight bearing was still painful. It occurred to the writer that if the feet could be supported with the toes off the ground, it would reduce the stress on the fractured metatarsals to a minimum, yet enable the patient to bear weight on the posterior part of the feet.

Two bath sponges, one for each foot, about 6 by 3 by $1\frac{1}{2}$ inches were shaped to fit the plantar surfaces of the feet and were held in place by figure-of-eight bandages, reinforced with adhesive strapping. The patient was comfortable, able to walk about the house, and, later, up and down stairs.

X-rays taken on January 26, 1938 showed firm exuberant callus formation around the fragment ends of the fractures of both feet. There was partial union of the fracture of the head and neck of the second right metatarsal with the same slight dorsal and inward angulation of the distal fragment. The fracture lines were faintly visible.

On January 29, 1938 the pads were removed and shoes padded within were substituted.

The patient returned to his regular occupation as a gasoline station attendant on February 14, 1938.

A NEW SUPRAPUBIC BLADDER EVACUATOR*

NELSE F. OCKERBLAD, M.D. AND HJALMAR E. CARLSON, M.D.

KANSAS CITY, MISSOURI

IN the operation of suprapubic cystotomy the operative field is often flooded by water and more or less infected urine when the bladder is opened. To

bladder is punctured which produces a water tight joint.

5. A pistol grip handle which allows the entire instrument to be manipulated with
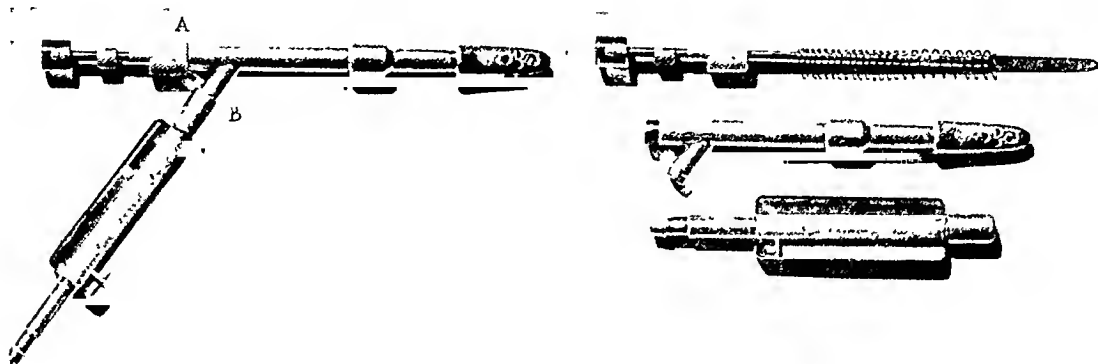
FIG. 1. Suprapubic bladder evacuator. A, assembled. B, taken apart for cleaning.

obviate contamination from this source various types of trocars are made, some of which have been designed for bladder surgery, although most of them are instruments designed for general surgical use. All of them have some defects, and most of them seem to fail at the crucial moment. Because we were not satisfied with the existing instruments we designed a suprapubic bladder evacuator which we have used and found quite satisfactory.

The principles of its construction are shown in Figure 1 and may be briefly summarized as follows:

1. A flat, adjustable knife blade which splits the bladder wall in a vertical direction, and is released by a spring as soon as the bladder wall is punctured.

2. An ovoid tip which follows the knife blade and rapidly divides the bladder wall.

3. Multiple openings in the tip which allow adequate suction even though the bladder wall may become adherent.

4. A raised collar against which the instrument is withdrawn as soon as the
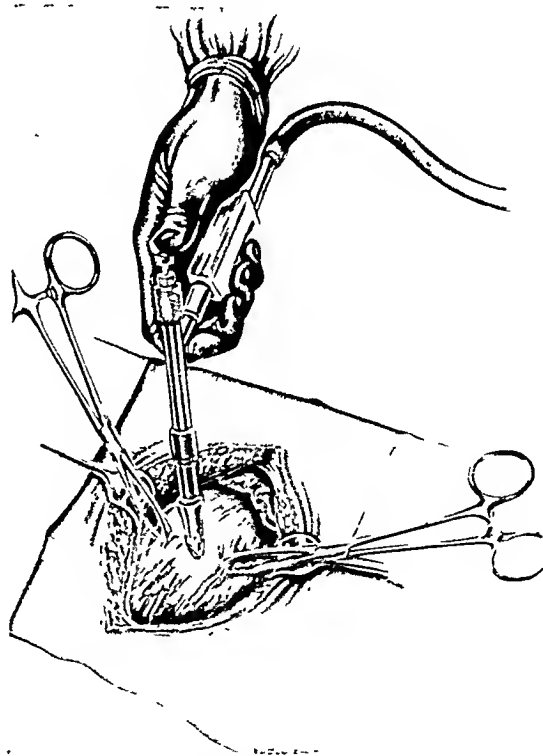
FIG. 2. Suprapubic bladder evacuator in position ready to puncture the bladder. Note that only one hand is necessary to manipulate the instrument.

one hand, including the thumb rest which operates the knife.

The instrument is easily dismantled for cleaning and oiling by taking it apart at points A and B. (Fig. 1.)

In using the instrument we have found that a well distended and exposed bladder, held firmly with two Allis clamps on either side of the point of intended puncture, gives sufficient resistance to secure a clean bladder opening. The active use of the instrument is illustrated in Figure 2. The bladder is rapidly evacuated when good suction is available, such as suction produced by a good water pump, or an anesthetist's pump which is kept in good repair. A non-collapsible rubber tubing should be used to insure prompt suction when the bladder is opened.

THE diuretic effect of the parathyroid hormone explains the polyuria and subsequent polydipsia of the hyperparathyroid patient which have been misinterpreted as signs of diabetes insipidus.

From—"The Endocrine Glands" by Max A. Goldzieher (Appleton-Century).

AN IMPROVED INSTRUMENT AND TECHNIQUE FOR INTESTINAL ANASTOMOSES*

JOHN R. O'SULLIVAN, M.D. AND BERNARD A. O'CONNOR M.D., F.A.C.S.

KEARNY, NEW JERSEY

THE instrument here presented in improved form consists of three elements: a U-shaped spreading device operated by a thumb-screw and two identical clamps set in clips at either end of the tension device.

In order to clarify the employment of the instruments, its application to the intestines in a routine operation is described. (Figs. 2 and 3.) The gastro-enterostomy, a typical example of enteroenterostomy, is used as a model.

The abdomen is prepared and the stomach approached through a high rectus incision. The desired folds of the stomach and jejunum are grasped with Allis tissue clamps placed about 5 inches apart. A piece of rubber dam tissue approximately 12 inches wide and 18 inches long is laid over the stomach and the long edge is applied to the fold of the stomach and held by Allis clamps. (Fig. 2.) The same procedure is followed with the jejunum.

The special small intestinal clamps are then fastened to the edges of the future anastomosis. The jaws of the clamp are opened by releasing the ratchet catch at the ends near the finger-rings. Thumbs and forefingers are inserted in the rings of the clamp, and the rubber-covered blade is placed in the center of the future anastomosis, with one side pressing against the stomach. The fold of intestine held by the Allis clamp is then gently drawn up between the center blade and the outside blade with the rubber insert. Pressure is brought on the ratchet catch so that it may securely retain the fold of the stomach. The loop of jejunum is guided into the space between the center blade and the

opposite rubber insert, and fastened in the same manner. This procedure is repeated with the fold of stomach and jejunum on the opposite side. In this way, not only are the surfaces of the bowel afforded protection by rubber tissue, but they are also securely held in place at two opposed points.

The clamps are set in the holding clips on either end of the tension device and spread apart by turning down the thumb-screw. (Fig. 2.) This action draws the loops of stomach and bowel into a straight, tense line, and facilitates suturing. Allis clamps are now removed. Simultaneously, the application of tension in the two folds of bowel extenuates the mucous membrane within and creates a sphincter-like action. Such tension limits the amount of intestinal contents that may escape upon the rubber tissue in use against the folds of the anastomosis and forms a watertight protection against contamination of the general peritoneal cavity.

The two loops of bowel are firmly held together and the posterior continuous peritoneal suture is applied. The stomach is incised along and directly through the edge of the fold and into the lumen. Active bleeding from arteries and large veins is controlled by clamp and ligature. The procedure is repeated with the jejunum.

At this point, the seromuscular stitch is used. The particular advantage of this device is that it enables one to turn corners of the seromuscular layer with neatness, and renders it water-tight simply by releasing the large tension clamp on the anastomosis while the corner is being turned. The seromuscular stitch is com-

pleted as usual and then the anterior peritoneal row of sutures is finished. The anastomosis is now complete.

out drainage. The general principles involved in anastomosing two loops of bowel together as outlined may be employed in
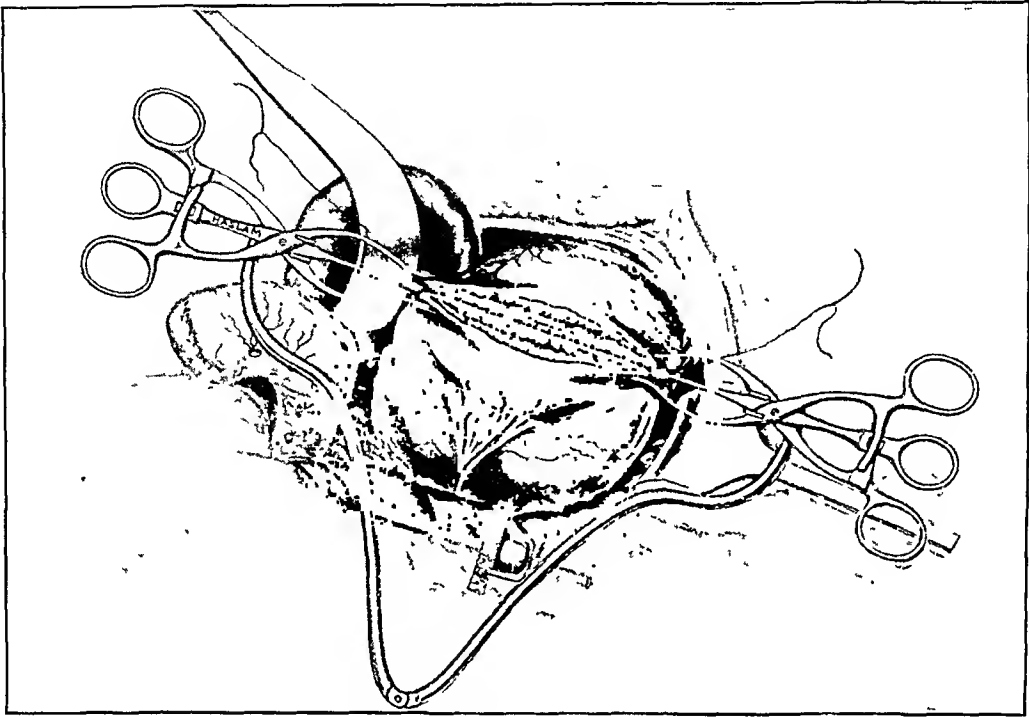
FIG. 1. The anastomosis of partial gastrectomy.
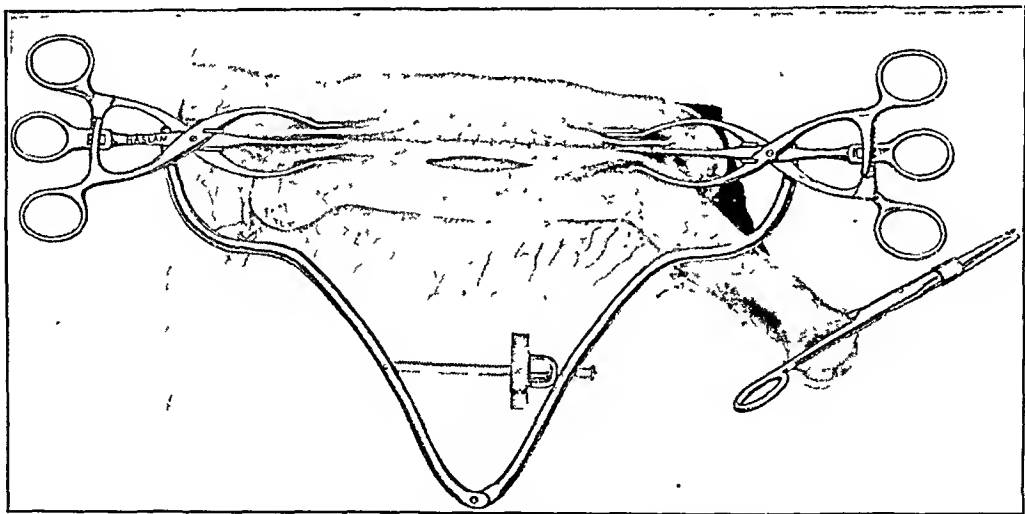
FIG. 2. Enteroenterostomy and/or gastroenterostomy.

The loops of the bowel may be wiped off with a mild aqueous solution of merthiolate. The gloves are changed, the clamps removed, and the rubber sheeting carefully lifted away. The abdomen may now be closed in layers in the usual manner with-

any type of intestinal side-tracking method which may be desired.

In dealing with the obstructive lesions of the gastrointestinal tract due to carcinoma, hernia, adhesions, volvulus, and intussusception in which a resection must

of necessity precede the anastomosis, the technique is modified as follows: The segment of diseased bowel is removed in

bleeding vessels is secured; (5) the stoma is protected.

The last point is particularly important
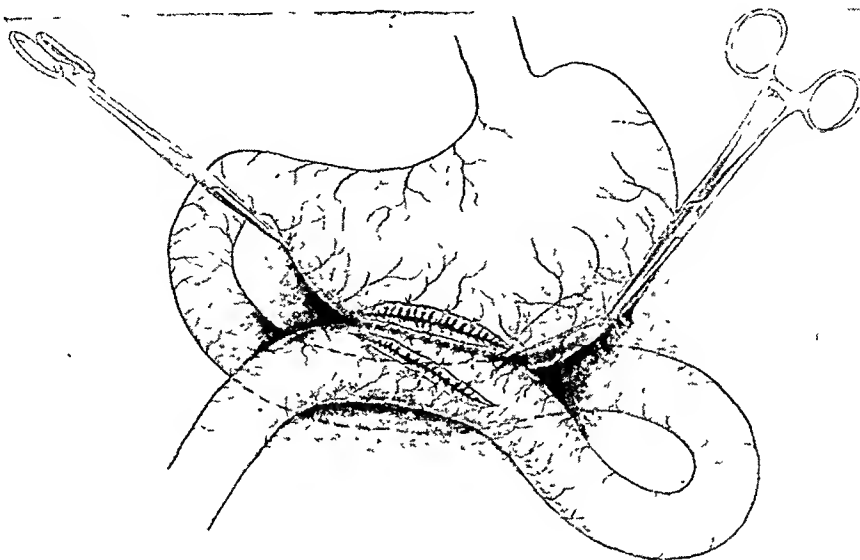
Fig. 3. Gastroenterostomy showing twisting of jejunal stoma from an axis parallel to gastric stoma.

the usual manner. Before the clamps on the severed edges of bowel are removed, the intestinal contents are aspirated through a medium-sized trochar inserted into the free edge of the bowel at that point, approximately the middle of the anastomosis. The ends are closed over and the isomesenteric edges of the bowel are laid together and joined as described. As an alternative method to prevent the intestinal contents from escaping, we cut off the sharp points of a tonsil forceps, thread the sides with rubber tubing, and clamp the forceps on the proximal loop of bowel about 5 inches above the anastomosis.

The instrument and technique offer the following advantages: (1) accessibility of deep anastomosis is enhanced, as the bowel segments are properly aligned and set in the tension device (Fig. 1); (2) control of the anastomosis is secured by an adjustable tension screw which is of particular importance in the use of the Connell suture for watertight closure of corners in gastric surgery; (3) soft rubber tubing, spring inserts and adjustable positions on the notches make for a non-traumatic grip (Fig. 2.); (4) primary ligation of active

in view of the fact that failure of the stoma to function in the first few days after operation is a serious and sometimes fatal complication. We consider such failure a result of edema, a tissue response to the injury inflicted on the intestinal tissues and blood vessels by the prolonged application of the heavy clamps now in use. Many poorly functioning stomas are due to a technical error at operation—failure to prevent rotation of the jejunum on its long axis. A flap-valve effect results, manifested clinically by intermittent vomiting some time after eating. We have found that this error may be prevented by suturing the various layers of the anastomosis under exactly regulated tension throughout the entire procedure. (Fig. 3.)

With individual variations in technique and the preparation of the patient, the clamps may be used in performing all types of gastric operations including the Billroth modifications. Enteroenterostomies and enterocolostomies may also be performed safely with a minimum of shock and hemorrhage when preceded by gastric lavage and cleansing enemas.

BOOKSHELF BROWSING

OBSTETRICS IN COLONIAL AMERICA*

CLAUDE EDWIN HEATON, M.D.

NEW YORK CITY

IN the new world, the seventeenth century was one of struggles and beginnings. The settlement of Jamestown in 1607, of Plymouth in 1620, and of New Netherlands in 1623, was accomplished only in the face of great suffering and privation. Frontier conditions existed everywhere. By the close of the century only three or four generations had been born along the Atlantic seaboard. The white race had secured a mere foothold on the Eastern landfall of a vast continent. In front was a wilderness and the lurking Indians, behind stretched three thousand miles of sea.

For the colonial mother the vicissitudes of a pioneer existence must have been especially trying. She established a home in a strange land with a rigorous climate. Children came in quick succession but many died during the first few weeks of life. Each year inevitably brought a new life and if the newborn was of the same sex as the one just lost he or she received his or her given name. Such names as Thankful and Patience were significantly common.

The care given to women in childbirth was almost entirely in the hands of women. The only reference found of a man taking part in midwifery during the century is to be noted in a letter written by Samuel Lee of New England in 1690 to Nehemiah Grew of London. He wrote: "I knew one Doctor Avery since deceased, a man of pretty ingenuity who from the ars veterinaria fell into some notable skill in physick and midwifery & invented some useful instrumts for that case." This was Doctor William Avery of Dedham and Boston who died March 18, 1687, at about 61 years of age. The instruments referred to were probably devised for the removal of a dead child since at that time the forceps were the family secret of the Chamberlens.

The names of many of the early colonial midwives have come down to us. Among these Anne Hutchinson's is the most notable. She was banished from Boston by the religious leaders because of radical views. She went to Rhode Island for a time and finally to Pelham in Westchester County where she and her family were massacred by the Indians during a raid. Her name is perpetuated in the Hutchinson River Parkway.

Ann Eliot, wife of the apostle to the Indians, practiced midwifery. Her tomb, erected by the town of Roxbury, Massachusetts in 1687, bears the legend, "She was thus honored for the great service she hath done this town." The grandson of the

---

606

apostle, Jared Eliot of Connecticut, was a noted pastor-physician famous for his sermons. One of these he preached on the death of his mother-in-law, Elizabeth, a midwife of Guilford. "She knew," he said, "when to exert herself vigorously and also when it was her strength to sit still."

In New Amsterdam midwifery was also in the hands of women. In 1660 the town council voted Hellegond Joris, town midwife, a salary of one hundred guilders ($40) a year for attending the poor of the city.

In the south each plantation had its own midwife. Negro midwives attended white women as well as negroes. In Surry County, Virginia, in 1685, the fee of Goodwife Thorpe, midwife, was 100 pounds of tobacco. Agnes Williams paid the Widow Hollens a dozen hens for attending her in confinement.

The only publication of any obstetrical significance printed in America during the seventeenth century was a reprint of a little book published in London in 1663. It illustrates exactly the sad state of midwifery at the period both here and abroad. The title reads: *A Present to be given to Teeming Women by their Husbands or Friends containing Scripture-Directions for Women with Child, how to prepare for the hour of Travel.* The author was John Oliver and the book was reprinted at Boston, by Benjamin Harris in 1694. Aside from some superficial advice to the pregnant woman on the care of her own health by avoiding excesses there is nothing of a practical nature. The bulk of the work deals with the duties of pregnant women which consisted of prayer, repentance, reading of scripture, meditation, resignation and preparation for death.

The genealogical records and old gravestone inscriptions bear mute testimony to the fact that childbirth in colonial America was accompanied by a frightful mortality for mothers and infants. Alice Morse Earle wrote: "It is heart-rending to read the entries in many an old family Bible—the records of suffering, distress and blasted hopes. There lies before me an old leather bound Bible with the records of my great grandfather's family. He had sixteen children. When the first child was a year and one half old the second child was born. The baby was but four days old when the older child died. Five times did that mother's heart bear a similar cruel loss when she had a baby in her arms; therefore, when she had been nine years married she had one living child, and five little graves bore record of her sorrow."

Many similar records could be found. Cotton Mather had fifteen children, only two of whom survived him. Judge Samuel Sewell recorded the birth of fifteen children in his famous diary. He buried two wives. The death of his daughter is recorded three days after the birth of her first child, the cause probably being infection.

The early midwives practiced internal podalic version, for Jane Hawkins, who delivered Mary Dyer of a monstrosity which caused a great disturbance among the New England witch hunters, testified in regard to the case that the baby "which was of ordinary bigness, came hiplings till she turned it."

An attempt was made to regulate the practice of medicine including obstetrics as early as 1649, in Massachusetts. A similar enactment was passed in New York in 1674. For the most part the treatment of the colonial mother was stark neglect or at best the meddlesome interference of ignorant grannies in whose hands she fared worse than the Indian squaw. Men's eyes were turned heavenward. The dangers of the invisible world loomed larger than the dangers of childbirth. Motherhood was just another tribulation in a sorrowful and suffering existence.

The eighteenth century brought great changes. Obstetrics in Europe advanced to a more scientific position than any other practical branch of medicine. The basic cause was a social evolution characterized by the growth of the democratic ideal and upsurge of the humanitarian spirit. The infant welfare movement emerged. When knowledge of the obstetric forceps became

available about 1773, physicians gained entrance into the lying-in chamber. They began to realize that to preserve infants more attention should be paid to the birth process. "In the nurture and management of infants as well as in the treatment of lying-in women," wrote Lettsom in his Medical Memoirs in 1774, "the reformation hath equalled that in the smallpox; by these two circumstances alone incredible numbers have been rescued from the grave."

The population of the colonies at the beginning of the eighteenth century was about 300,000. By the middle of the century enough capital had been accumulated and enough leisure acquired to enable a fairly large group of young colonials to cross the seas and contact the main current of medical learning at Leyden and London and Edinburgh. They brought back a leaven which accelerated the rapid rise of American medicine. Among those who went abroad to study were James Lloyd, the earliest practitioner in New England to specialize in midwifery. He studied under William Hunter and William Smellie. Lloyd was a great teacher of obstetrics and had as his pupils in Boston such men as Oliver Prescott, Isaac Rand and Marshall Spring. That the efforts of Lloyd and his pupils to establish obstetrics as a branch of medical practice met with success may be inferred from the following advertisement from the Boston Evening Post of November 10, 1781:

"The physicians of the town of Boston, hereby inform the public, that, in consideration of the great fatigue and inevitable injury to their constitution, in the practice of midwifery, as well as the necessary interruption of other branches of their profession, they shall, for the future, expect that in calls of this kind, the fee be immediately discharged."

The most notable student of Hunter and Smellie was William Shippen, Jr., who returned to Philadelphia in 1762 and immediately announced a course of lectures which included a few plain general directions in the study and practice of midwifery. The next year he gave a full course of lectures on obstetrics, the first in America. Philadelphia took the lead in obstetrics for a great many years. At the Philadelphia Alms House was established what Agnew considers the first obstetrical clinic. As early as 1770 and possibly earlier, Dr. Bond and Dr. Evans personally instructed students who were allowed to attend cases of labor. At the Pennsylvania Hospital Dr. Bond commenced clinical lectures upon midwifery as early as 1766 but the lying-in ward was not opened until 1807.

In New York City, the first professorship devoted solely to obstetrics was established in 1767 at Kings College (later Columbia), with J. V. B. Tennent as the first professor. In Philadelphia, Shippen held the combined chair of Surgery, Anatomy and Obstetrics in the University of Pennsylvania in 1792. There was no chair of obstetrics at Harvard University until 1815.

After the Revolutionary War Samuel Bard gave a private course of lectures on obstetrics, the medical school being disorganized. In 1799 a lying-in ward was established in the Almshouse of the city of New York. The same year Valentine Seaman gave lectures at the Almshouse on midwifery, to women exclusively. The fact has never been brought out that the earliest clinical teaching of medicine in New York City was given at the Almshouse which later became Bellevue Hospital. The New York Lying-In Hospital began as a direct result of the terrible mortality from the yellow fever in the summer of 1798 "when many women were bereaft of their husbands." David Hosack was responsible for the establishment of the hospital which was incorporated in 1799.

In 1789, George Buchanan of Baltimore delivered a course of lectures on the diseases of women and children to a class of nine students and at the same period Andrew Wiesenthal lectured at

his own house on anatomy, physiology, pathology, operative surgery and the gravid uterus. During the succeeding winter Dr. Buchanan also delivered a course of lectures in midwifery. Buchanan attempted to found a lying-in hospital but failed.

The obstetrical literature of the period was, of course, derived from the teachings of the great British obstetricians of the eighteenth century. In 1786, *An Abridgement of the Practice of Midwifery and a set of Anatomical Tables* by William Smellie was reprinted in Boston. This work was the first illustrated medical book we have and also contains the first illustration of the obstetrical forceps to appear in this country.

Another great classic reprinted here in 1793 was Charles White's *Treatise on the Management of Pregnant and Lying-in Women.* Excellent prophylactic rules for puerperal fever were laid down. White instituted the principle of uterine drainage by placing his patients in a sitting position shortly after delivery. He was an advocate of antiseptic injections into the uterus for the removal of putrid matter. He taught the necessity of absolute cleanliness in the lying-in chamber and urged the isolation of infected patients.

A second type of literature appearing at the time should not be overlooked, namely, student theses. Space will permit the discussion of only one—*A Dissertation on the Puerperal Fever* delivered by Peter de Sales la Terriere, a Canadian student at Harvard. This was printed in Boston in 1789. Significant is his statement that: "The numbers cut off by this disease in Canada have moved my compassion." Discussing treatment he speaks of "An ingenious practitioner of eminence in this commonwealth" who "recommended an antiseptic course," and who "under appearances actually putrid prescribed antiseptics."

The earliest contributions to periodical literature were of necessity printed in London publications. Early operations for advanced extra-uterine pregnancy were reported by John Bard in 1762 and Charles

McKnight in 1795. An early report, hitherto overlooked by historians, was made by William Baynham of Virginia in 1791 and published in the first volume of *Medical Facts and Observations*, London, 1791.

In the closing years of the century a number of contributions on obstetrical subjects were printed in this country in works of a periodical nature. Leverett Hubbard contributed a paper on deformed fetus to the first volume of *Transactions*, published by the Medical Society of New Haven County in 1788.

The medical papers communicated to the Massachusetts Medical Society contained at least seven contributions on obstetrics. Tragic indeed is *An Account of a Case of Ruptured Uterus* by Oliver Prescott. The patient had regular pains for eighty-two hours. The head was unengaged, for the pelvis was contracted. Several doctors were in attendance to attempt instrumental delivery when the cervix became dilated, but before this occurred the woman died. Autopsy revealed a ruptured uterus.

More courageous was Jesse Bennet, a country practitioner in the Valley of Virginia, in the case of his own wife. He performed cesarean section, saving mother and child. This was the first successful cesarean in America.

Dr. Thomas Archer of Harfordtown, Maryland, reported a singular case of difficult parturition successfully treated in 1797. He had watched the case for a long time and labor failed to progress. Finally he decided to act. We quote his words: "Unknown to the patient or any of the attendants except the midwife, who held the candle (for it was now night), with a common spear-pointed lancet, I made three incisions in the neck of the womb, which was very much distended; each about two inches in length, viz., one from the uterus leading toward the urethra, one toward the perineum; and the other toward the labium." The patient was then delivered and was up and about three weeks after.

At the close of the colonial period considerable progress had been made in obstetrics. The religious and mystical phase of the seventeenth century had given way to a more rational and objective approach. No great contributions had been made, but highly important beginnings. Obstetrics had found a place in two medical colleges. Lying-in wards had been opened and some clinical teaching commenced by men with really excellent training. Classic British works on obstetrics had been reprinted.

The forceps had been introduced. Contributions by American physicians had begun to appear. Cesarean section had been successfully performed as well as several operations for extra-uterine gestation. This advance was to be accelerated in the nineteenth century and prior to the introduction of asepsis and antisepsis carry with it a tremendous price in the ravages of puerperal infection. Nevertheless, the era of blistering, purging and blood-letting was passing away.

THE most striking new fact clearly demonstrated in respect of the urinary excretion of prolan (in pregnancy) is that an enormous excretion is observed about the 50-60 day following the last menstrual period . . . The highest figures reported are 100,000 to 300,000 rat units per liter of urine in comparison with a later fall to about 3,000 rat units per liter. From—"The Physiology and Pharmacology of the Pituitary Body" by H. B. Van Dyke (University of Chicago Press).

AUTHOR INDEX TO VOLUME XLV

SUBJECT INDEX TO VOLUME XLV

(Bo. B) = Bookshelf Browsing; (E.) = Editorial